APPENDIX A

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

APPENDIX A

PHYSICAL AND CHEMICAL PROPERTIES of CDDs AND CDFs

The chemical structure and nomenclature of CDDs and CDFs are discussed in the Introduction (Chapter 1). The following discussion is referenced to material presented in Table A.1 of this Appendix.

COMPOUND AND MOLECULAR FORMULA

Of the 75 CDD congeners and 135 CDF congeners, only 28 CDD and 3 CDF congeners have been found to have experimentally derived data on physical and chemical properties. The lack of adequate identification and characterization of CDD and CDF compounds stems largely from analytical problems (Monitoring Chapter).

CAS NUMBER

The Chemical Abstract Service (CAS) numbers are from the publication "Registry of Toxic Effects of Chemical Substances" (NIOSH, 1983). Each CAS number in Table A.1 identifies a specific compound. This unique number, with the aid of a computer, gives the reader rapid access to toxicity and chemical information necessary for the preparation of safety measures or hazard evaluations for these substances.

MOLECULAR WEIGHT

As the number of chlorine atoms in a compound increases, the molecular weight of that compound increases correspondingly. MonoCDD, with only one chlorine atom, weighs 218.64 grams/-mole, whereas octaCDD, with eight chlorine atoms, weighs 459.72 grams/mole.

PHYSICAL STATE

At standard temperature and pressure CDDs are in solid, colorless form, usually appearing in the shape of crystals or needles. The physical states of CDFs have not been adequately described in the literature.

MELTING POINT

The CDDs and CDFs are considered to be very stable and resistant towards heat. The less chlorinated CDD compounds begin to melt at 80° to 90°C, whereas the more highly chlorinated compounds generally melt above 200°C.

VAPOR PRESSURE

The literature gives a wide range of vapor pressure values. Values given for 2,3,7,8-tetraCDD are: 1.5×10^{-7} mm Hg (25°C)

TABLE A.1 (continued)

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
Page 2								
CDDs								
1,2,3,4-tetraCDD $C_{12}H_4Cl_4O_2$	30746-58-8	321.96	Colorless Needles	188-190	N/A	8.8	N/A	Sarna et al., 1984; Pohland and Yang 1972; NIOSH, 1983
1,2,3,8-tetraCDD $C_{12}H_4Cl_4O_2$	53555-02-5	321.96	N/A	N/A	N/A	N/A	N/A	NIOSH, 1983
1,3,6,8-tetraCDD $C_{12}H_4Cl_4O_2$[b]	33423-92-6	321.96	Colorless Needles	219-219.5	N/A	9.0-9.26	0.353	Sarna et al., 1984; Pohland and Yang, 1972; NIOSH, 1983; Muir et al., 1985a
1,3,7,8-tetraCDD $C_{12}H_4Cl_4O_2$	50585-46-1	321.96	N/A	193.5-195	N/A	N/A	N/A	Kende et al., 1974; NIOSH, 1983
2,3,6,7-tetraCDD $C_{12}H_4Cl_4O_2$	34816-53-0	321.96	N/A	N/A	N/A	N/A	N/A	NIOSH, 1983
2,3,7,8-tetraCDD $C_{12}H_4Cl_4O_2$	1746-01-6	321.96	Colorless Needles, Crystalline	305-306	1.7×10^{-6} to 1.5×10^{-9}	6.15	0.2	Pohland and Yang, 1972 U.S. EPA, 1978, 1984a, 1985b; NIOSH, 1983; Esposito et al., 1980
pentaCDD (isomer unspecified) $C_{12}H_3Cl_5O_2$	N/A	356.4	N/A	N/A	N/A	6.8 at 25^{o}C	.04 at 25^{o}C	U.S. EPA, 1985b
1,2,3,4,7-pentaCDD $C_{12}H_3Cl_5O_2$	N/A	356.4	Colorless Solid	195-196	N/A	8.64-9.7	.132	Pohland and Yang, 1972; Sarna et al., 1984; NIOSH, 1983; Muir et al., 1985a
1,2,3,7,8-pentaCDD $C_{12}H_3Cl_5O_2$	40321-76-4	356.4	N/A	240-241	N/A	N/A	N/A	NIOSH, 1983 Gray et al., 1976

TABLE A.1

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
CDDs								
monoCDD $C_{12}H_7ClO_2$	39227-53-7	218.64	Colorless Crystals	80-90	N/A[d]	N/A	N/A	NIOSH, 1983
diCDD $C_{12}H_6Cl_2O_2$	39227-54-8	253.08	Colorless Solid	88-89	N/A	5.6	N/A	Sarna, et al., 1984; NIOSH, 1983; Pohland and Yang, 1972
1,3-diCDD $C_{12}H_6Cl_2O_2$	50585-39-2	253.08	Colorless Solid	113.5 - 114.5	N/A	N/A	N/A	Kende et al., 1974; NIOSH, 1983
1,6-diCDD $C_{12}H_6Cl_2O_2$	38178-38-0	253.08	Colorless Needles	N/A	N/A	N/A	N/A	NIOSH, 1983
2,3-diCDD $C_{12}H_6Cl_2O_2$	29446-15-9	253.08	Colorless Solid	163 - 164	N/A	N/A	N/A	Pohland and Yang, 1972; NIOSH, 1983
2,7-diCDD $C_{12}H_6Cl_2O_2$	33857-26-0	253.08	Colorless Crystals	209 - 210	7.0×10^{-6}	6.5	N/A	Sarna et al., 1984; NIOSH, 1983; Pohland and Yang, 1972
2,8-diCDD $C_{12}H_6Cl_2O_2$	38964-22-6	253.08	Colorless Solid	143 - 150	6.8×10^{-6}	N/A	N/A	Pohland and Yang, 1972; NIOSH, 1983
1,2,4-tri-CDD $C_{12}H_5Cl_3O_2$	39227-58-2	287.52	Colorless Solid	128-129	N/A	7.6	N/A	Pohland and Yang, 1972; NIOSH, 1983; Sarna et al., 1984
2,3,7-tri-CDD $C_{12}H_5Cl_3O_2$	33857-28-2	287.52	N/A	157-158	3.6×10^{-6}	N/A	N/A	Gray et al., 1976; Kende et al., 1974; NIOSH, 1983 U.S. EPA, 1978

TABLE A.1 (continued)

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature(^{o}C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
CDDs								
octaCDD $C_{12}Cl_8O_2$	3268-87-9	459.72	N/A	330	1.8×10^{-7}	10.07 12.6	.0004	NIOSH, 1983; Pohland and Yang, 1972; Muir et al., 1985a; U.S. EPA, 1978
CDFs[c]								
monoCDF $C_{12}H_7ClO$	N/A	202.42	N/A	N/A	N/A	N/A	N/A	Sarna et al., 1984
diCDF $C_{12}H_6Cl_2O$	43047-99-0	237.08	N/A	N/A	7.0×10^{-6}	N/A	N/A	NIOSH, 1983; U.S. EPA, 1978
2,3,7,8-tetraCDF $C_{12}H_4Cl_4O$	51207-31-9	305.96	N/A	N/A	2.0×10^{-6}	N/A	N/A	NIOSH, 1983; U.S. EPA, 1978

a) All K_{ow} values are averaged values of experimentally derived quantities using reversed-phase HPLC within each laboratory.

b) 1,3,6,8-tetraCDD Henry's Constant is 6.81×10^{-5} atm $m^3/^{o}k$ mol (Pohland and Yang, 1972).

c) Information concerning CDFs is very limited.

d) N/A = "not available".

TABLE A.1 (continued)

PHYSICAL AND CHEMICAL CHARACTERISTICS OF CDDs AND CDFs

Compound/ Molecular Formula	CAS Number	Molecular Weight (grams/ mole)	Physical State (at Standard Temperature($^{\circ}$C) and Pressure)	Melting Point	Vapor Pressure (mmHg)	Log K_{ow} (Octanol/ Water Partition Coefficient[a])	Solubility in Water (ug/l)	Reference
Page 3								
CDDs								
1,2,4,7,8-pentaCDD $C_{12}H_3Cl_5O_2$	58802-08-7	356.4	N/A	N/A	N/A	N/A	N/A	NIOSH, 1983; Pohland and Yang, 1972
hexaCDD (isomer unspecified) $C_{12}H_2Cl_6O_2$	N/A	N/A	N/A	N/A	N/A	7.6 at 25°C	.008 at 25°C	U.S. EPA, 1985b
1,2,3,4,7,8-hexaCDD $C_{12}H_2Cl_6O_2$	39227-28-6	390.84	Colorless Solid	275	N/A	9.19 10.5	N/A	Pohland and Yang, 1972; Sarna et al., 1984; NIOSH, 1983; Muir et al., 1985a
1,2,3,6,7,8-hexaCDD $C_{12}H_2Cl_6O_2$	34465-46-8	390.84	N/A	285-286	N/A	N/A	N/A	Gray et al., 1976; NIOSH, 1983
1,2,3,6,7,9-hexaCDD $C_{12}H_2Cl_6O_2$	N/A	390.84	N/A	N/A	N/A	N/A	N/A	Pohland and Yang, 1972;
1,2,3,7,8,9-hexaCDD $C_{12}H_2Cl_6O_2$	19408-74-3	390.84	N/A	243-244	N/A	N/A	N/A	Gray et al., 1975; NIOSH, 1983
1,2,4,6,7,9-hexaCDD $C_{12}H_2Cl_6O_2$	N/A	390.84	Colorless Solid	238-240	6.6×10^{-7}	N/A	N/A	Pohland and Yang, 1972; U.S. EPA, 1978
1,2,3,4,6,7,8-heptaCDD $C_{12}HCl_7O_2$	35822-46-9	390.84	N/A	N/A	N/A	11.5	N/A	NIOSH, 1983; Sarna et al., 1984
1,2,3,4,6,7,9-heptaCDD $C_{12}HCl_7O_2$	N/A	390.84	N/A	N/A	3.0×10^{-7}	N/A	N/A	NIOSH, 1983; U.S. EPA, 1978

APPENDIX B

SOURCES OF CDDs AND CDFs

Freeman and Schroy, 1986), 5.6×10^{-7} ($25^{\circ}C$) (Podoll, 1986) and 1.7×10^{-6} (estimated at $25^{\circ}C$) (U.S. EPA, 1978). These are low vapor pressures, generally representing low volatility. However, Freeman and Schroy (1986) noted that DDT, which has a similar vapor pressure of 1.4×10^{-7} is known to volatilize readily from soil and water.

LOG K_{OW} (LOGARITHM OF OCTANOL/WATER PARTITION COEFFICIENT)

This coefficient measures the partitioning of a compound into the two phases of an octanol-water mixture, indicating the compound's relative concentration in these two solvents. The partition coefficients of CDDs and CDFs are relatively high (i.e., the compounds preferentially dissolve in octanol and lipid) and generally increase with increasing chlorination. In lieu of actual field data, the partition coefficients are frequently used as indicators for potential concentration in biota.

SOLUBILITY IN WATER AND OTHER SOLVENTS

In general, CDDs and CDFs have extremely low water solubilities, are only slightly soluble in most organic solvents such as acetone, but are more soluble in others. The available data suggest that the less chlorinated compounds (i.e., diCDD and triCDD) are more soluble in aliphatic solvents (i.e., acetone, methanol) whereas the more highly chlorinated compounds are more soluble in aromatic hydrocarbon solvents. CDDs and CDFs are insoluble in dilute alkali, although the more highly chlorinated compounds (i.e., heptaCDD and octaCDD) are "degraded" by a few minutes' boiling with aqueous-alcoholic potassium hydroxide (Crosby, 1981). The solubility of 2,3,7,8-tetraCDD and octaCDD in various solvents is shown in Table A.2.

TABLE A.2

SOLUBILITY OF 2,3,7,8-TETRACDD AND OCTACDD IN VARIOUS SOLVENTS AT $25^{\circ}C$

Solvent	Solubility (mg/l) 2,3,7,8-tetraCCD	octaCDD
o-dichlorobenzene	1400	1830
chlorobenzene	720	---
anisole	---	1730
xylene	---	3580
benzene	570	---
chloroform	370	560
n-octanol	48	---
methanol	10	---
acetone	11	---
dioxane	---	380
water	0.0002	---

Source: Esposito et al., 1980

APPENDIX B

SOURCES OF CDDs AND CDFs

The CDD and CDF toxicity has been brought to public attention through media coverage of several major incidents: the chemical plant accident in Seveso, Italy in 1976; the fire in the Binghamton, N.Y. State Office Building in 1981; the poisonings at horse arenas in Missouri in 1971 and in Times Beach, MO, in 1982-83; the Yusho disease in Japan in 1968 and in Taiwan in 1979; and the herbicide spraying program in Vietnam in the late 1960's (Rappe, 1984).

CDDs and CDFs are not intentionally produced, except for the synthesis of analytical standards. Rather, they are found as impurities in a variety of commercial products like chlorinated phenols and their derivatives, chlorinated diphenyl ethers and polychlorinated biphenyls. They are also formed through the combustion of certain chlorinated hydrocarbons. A description of these and other sources of CDDs and CDFs follows:

PHENOXY HERBICIDES

Concentrations of .02 to 54 ug/g of 2,3,7,8-tetraCDD have been found in drums of the phenoxy herbicide 2,4,5-trichlorophenoxy acetic acid (more commonly called Agent Orange or 2,4,5-T) (Firestone, 1978; Esposito et al., 1980).

The parent compound of 2,4,5-T is 2,4,5-trichlorophenol. From 2,4,5-trichlorophenol, several other herbicides, including Silvex, are derived (U. S. EPA, 1985c). It should be pointed out that the occurrence of 2,3,7,8-tetraCDD in the environment can be mainly related to the synthesis of 2,4,5-trichlorophenol and the use of products prepared from this compound or incineration reactions. The occurrence of other CDDs and CDFs can be related to the synthesis and use of a variety of other products (WHO, 1985).

HEXACHLOROPHENE

The bactericide, hexachlorophene, also is prepared from 2,4,5-trichlorophenol. Samples from one study showed concentrations of 0.2 to 0.5 ug/g of 2,3,7,8-tetraCDD (Baughman, 1974).

CHLOROPHENOLS

Chlorophenols are commercially made either by direct chlorination of phenol or by hydrolysis of chlorobenzenes, with the process dependent on the compound desired. Chlorination of phenols yields 2,4-dichlorophenol, 2,4,6-trichlorophenol, 2,3,4,6-tetrachlorophenol, or pentachlorophenol (PCP). Hydrolysis of chlorobenzenes is used mainly for the production of 2,4,5-trichlorophenol and PCP (Nilsson, et al., 1978).

TABLE B.2

CDF CONCENTRATIONS IN COMMERCIAL PCBs (ug/g)
(Bowes et al., 1975)

Sample	Tetra	Penta	Hexa	Total
Aroclor 1248 (1969)	0.5	1.2	0.3	2.0
Aroclor 1254 (1969)	0.1	0.2	1.4	1.7
Aroclor 1254 (1970)	0.2	0.4	0.9	1.5
Aroclor 1260 (1969)	0.1	0.4	0.5	1.0
Aroclor 1260 (lot AK3)	0.2	0.3	0.3	0.8
Aroclor 1016 (1972)	ND	ND	ND	--
Clophen A 60	1.4	5.0	2.2	8.4
Phenoclor DP-6	0.7	10.0	2.9	13.6

ND = Not detected

DIPHENYL ETHER HERBICIDES

Yamagishi, et al. (1981) found CDDs and CDFs in the commercial diphenyl ether herbicides, CNP, NIP, and X-52. The major tetraCDDs identified were 1,3,6,8 and 1,3,7,9-isomers. No 2,3,7,8-tetraCDD could be found in these samples. Table B.3 summarizes the results of this study:

TABLE B.3

CDD AND CDF CONCENTRATIONS IN COMMERCIAL
DIPHENYL ETHER HERBICIDES (ug/g)

(Yamagishi et al., 1981)

	CNP	NIP	X-52
TriCCDs	ND	0.15	0.03
TetraCDDs	14.0	0.38	0.03
PentaCDDs	37	0.05	0.01
HexaCDDs	0.8	ND	ND
MonoCDFs	ND	0.34	0.48
DiCDFs	0.35	0.12	0.21
TriCDFs	0.41	0.47	0.45
TetraCDFs	0.4	0.29	0.32
PentaCDFs	1.0	ND	0.08
HexaCDFs	0.2	ND	ND

ND = Not detected

Chlorophenols have been used extensively in the wood industry as fungicides, bactericides, slimicides, and mold inhibitors. The most important use of 2,4,6-tri, 2,3,4,6-tetra, and pentachlorophenols (or their salts) is for wood protection and preservation against fungal damage. Chlorophenols contain a variety of contaminants including CDDs and CDFs, as in Table B.1 following:

TABLE B.1

CDD AND CDF CONCENTRATIONS IN COMMERCIAL CHLOROPHENOLS (ug/g)
(RAPPE et al., 1979)

	2,4,6-trichloro-phenol	2,3,4,6-tetrachloro-phenol	PCP[a/]	PCP
TetraCDDs	<0.1	<0.1	<0.1	<0.1
PentaCDDs	<0.1	<0.1	<0.1	<0.1
HexaCDDs	<1	<1	<1	2.5
HeptaCDDs	<1	10	0.5	175
OctaCDD	<1	2	4.3	500
TetraCDFs	1.5	0.5	<0.1	<0.1
PentaCDFs	17.5	10	<0.1	<0.1
HexaCDFs	36	70	0.03	<0.3
HeptaCDFs	4.8	70	0.5	19
OctaCDF	<1	10	1.1	25

[a/] Purified product

POLYCHLORINATED BIPHENYLS (PCBs)

Bowes et al., (1975), examined PCB formulations produced in the United States (Aroclor), France (Phenoclor), and Germany (Clophen). They reported that the most abundant CDFs had the same retention time as 2,3,7,8-tetraCDF and 2,3,4,7,8-pentaCDF. Quantitative results, reported as isomer group concentrations of CDFs in commercial PCBs, are given in Table B.2.

TABLE B.4 (continued)

POTENTIAL CDD AND CDF SOURCES IN CALIFORNIA
(CARB AND CDHS, 1986)

Source Category	Operational in Calif.	Proposed for Calif.	Estimate of Relative Emissions[1/]
Area Sources:			
Mobile Sources	NA		Unknown
Wood Stove/Fireplaces	NA		Unknown
Forest Fire/Agricultural Burning	NA		Unknown

ND - no data
NA - not applicable

1/ This is an estimate of the expected emissions relative to the other sources listed.

2/ Statewide number estimated from data supplied by San Diego Air Pollution Control District and the South Coast Air Quality Management District.

3/ When burning wood treated with chlorophenol; otherwise sawmills are rated as "low".

4/ Most sawmills have the capability to incinerate some or all of the woodwaste produced at the facility. A wood/bark boiler may be used at a a sawmill to incinerate wastes. This source category may overlap other source categories listed in the table.

FLY ASH AND COMBUSTION PRODUCTS

Combustion sources of CDDs and CDFs have only been studied for a relatively short period of time. It is believed that CDDs and CDFs adsorb onto airborne particles which are eventually deposited on soil and water. According to a recent California Air Resources Board (CARB) report (CARB and CDHS, 1986), emissions of CDDs and CDFs from combustion sources into the atmosphere appear to be the major environmental source of dioxins. Few potential sources, except for municipal waste resource recovery facilities, have been adequately tested. Based on tests of municipal waste resource recovery facilities of the type expected to be built in California, CARB estimates that 18 to 308 pounds of CDDs and 41 to 663 pounds of CDFs would be emitted in California annually if all currently proposed facilities (39) are constructed (CARB, 1986). Table B.4 identifies potential CDD and CDF sources in California:

TABLE B.4

POTENTIAL CDD AND CDF SOURCES IN CALIFORNIA
(CARB AND CDHS, 1986)

Source Category	Operational in Calif.	Proposed for Calif.	Estimate of Relative Emissions[1]
Point Sources:			
Municipal Waste Incinerators and Refuse Derived Boilers	1	35	High
Commercial Waste Oil Burners	30+	ND	Unknown
Hazardous Waste Incinerators	17	3	Low
Industrial Boilers Cofiring Wastes	0	0	Unknown
Wire Reclamation Incinerators	76[2]	ND	Unknown
Sewage Sludge Incinerators	8	ND	Unknown
Wood/Bark Boilers	59	ND	High[3]
Black Liquor Boiler	4	0	Unknown
PCP Sludge Incinerators	ND	ND	High
Cement Kilns Cofiring Wastes	1	1	Low
Hospital Incinerators	311[2]	ND	Unknown
Sawmills[4]	86	ND	High[3]

TABLE B.6

FORMATION OF CDDs AND CDFs BY THERMAL PROCESSES
(Rappe, 1984)

Starting Material	Thermal Process	Product
2,4,5-T salt	Pyrolysis	2,3,7,8,-tetraCDD
2,4,5-T (on vegetation)	Pyrolysis	No tetraCDD
	Burning	No tetraCDD
Chlorophenate	Burning	CDDs[a] + CDFs
Polychlorinated biphenyls	Pyrolysis	CDFs[b]
Polychlorobenzenes	Pyrolysis	CDFs + CDDs[c]
Chlorodiphenyl ethers	Pyrolysis	CDFs + CDDs
Polyvinylchloride	Pyrolysis	Polychlorobenzenes

[a] CDDs formed by dimerization and a nonspecific dechlorination

[b] Other products: hexa- and pentachlorobenzenes

[c] Other products: PCBs, polychlorinated naphthalenes

Combustion sources believed to have the greatest potential to emit CDDs have been identified by U.S. EPA (1984b) and are presented in Tables B.5 and B.6.

TABLE B.5

COMBUSTION SOURCES BELIEVED TO HAVE THE GREATEST POTENTIAL TO EMIT CDDs (U.S. EPA, 1984b)

Source	Rationale
Municipal Waste Incinerators	TetraCDD[1/] Detected
Refuse Derived Fuel[2/] Boilers	TetraCDD Detected
Commercial Waste Oil Burners	TetraCDD Detected
Hazardous Waste Incinerators	TetraCDD Detected
Industrial Boilers Cofiring Wastes	TetraCDD Detected
Wire Reclamation Incinerators	TetraCDD Detected
Pentachlorophenol Sludge Incinerators	TetraCDD Detected
Sewage Sludge Incinerators	CDD[2/] Detected
Mobile Sources	TetraCDD Detected
Wood Stove/Fireplaces	TetraCDD Detected
Wood/Bark Boilers	Experimental results with pentachlorophenol-treated wood
Black Liquor Boilers	Elevated polycyclic organic matter in effluent
Cement/Lime Kilns Cofiring Wastes	Precursors present
Hospital Incinerators	Burn plastics, equipped with low stacks and are located in urban areas
Forest/Grass/Agricultural Burning	Areas where chlorinated pesticides have been applied

1/ TetraCDD = tetrachlorodibenzodioxin. Available analyses are mixed, with some researchers reporting "total tetras" and others reporting 2,3,7,8-tetraCDD or both. The presence of tetraCDDs generally indicates some likelihood of 2,3,7,8-tetraCDD being present.

2/ CDD = Total of all chlorinated dibenzodioxin congeners. While detection of CDDs does not necessarily indicate presence of tetraCDD or 2,3,7,8-tetraCDD, there are sufficient data to infer such in this case.

APPENDIX C

BACKGROUND DOCUMENTS FOR CHAPTER 4: MAMMALIAN TOXICOLOGY

CHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS IN CHLOROPHENOL WOOD PRESERVATIVES;CALIFORNIA HAZARD ASSESSMENT

COMMENTS OF THE DOW CHEMICAL COMPANY

These comments have been prepared by Dr. R. J. Kociba in response to the draft document dated September 26, 1986, and entitled, "Chlorinated Dibenzo-p-dioxins and Dibenzofurans in Chlorophenol Wood Preservatives; California Hazard Assessment." This draft document has been prepared by the California State Water Resources Control Board, Division of Water Quality, Sacramento, California.

Due to the limited time available for our review of this draft document, our comments will be limited to the following issues: 1) mechanism of animal carcinogenicity for 2,3,7,8 TCDD; 2) interspecies sensitivity of man to dioxin relative to the laboratory animal species used in chronic toxicity studies; and 3) use of toxicity equivalent factors (TEF) in dealing with mixtures of other dioxins and furans relative to the 2,3,7,8 TCDD.

As this draft document has drawn heavily from previous documents on dioxin such as the California Air Resources Board Report, 1986, U.S. EPA Health Assessment Document, 1985, U.S. EPA Ambient Water Quality Criterion Document for 2,3,7,8 TCDD, 1984, as the underlying source of the data (rather than the original scientific literature data) we are also attaching the pertinent comments that we submitted previously in response to comments on drafts of the U.S. EPA Health Assessment Document (Attachment A), The U.S. EPA Water Quality Criteria Document (Attachment B) and the California Air Resources Board Report (Attachment C).

These comments made previously on these other documents are intended to supplement the specific comments given below.

COMMENTS ON MECHANISM OF ANIMAL CARCINOGENICITY FOR 2,3,7,8 TCDD:

Analysis of the data available indicates that the 2,3,7,8 TCDD (as well as other chlorinated dioxins and furans) does (do) not possess any significant mutagenic/clastogenic potential. Other studies have reported a lack of covalent bonding of 2,3,7,8 TCDD to DNA or RNA, with maximal binding potential 4-6 orders of magnitude less than that observed with carcinogens. Likewise, 2,3,7,8 TCDD does not stimulate unscheduled DNA synthesis when tested in rat hepatocytes or in a human cell line. The various studies with TCDD in regard to tumor initiation, promotion and cocarcinogenesis indicate that on the mechanistic basis TCDD is more correctly categorized as a tumor promoter rather than an initiator. This categorization of 2,3,7,8 TCDD as a promoter has been the basis upon which at least four agencies/expert panels have recently adopted a threshold basis for extrapolation from the chronic animal bioassays for the derivation of lifetime human exposure control recommendations for 2,3,7,8 TCDD.

HEALTH EFFECTS OF 2,3,7,8-TETRACHLORODIBENZO-p-DIOXIN AND RELATED COMPOUNDS

RESPONSE TO PUBLIC COMMENTS

Prepared by

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25 September 1985

Attachment D is a copy of a paper currently in press that addresses the mechanistic basis for using a threshold basis of extrapolation from the animal studies to man.

COMMENTS ON INTERSPECIES SENSITIVITY OF MAN TO DIOXIN RELATIVE TO LABORATORY ANIMALS:

The publication of Roberts, Shear and Okey (Attachment E) reports on the relative levels of the receptor binding protein for 2,3,7,8 TCDD in the tissues of human relative to the levels in tissues of laboratory animals. These data indicate that human tissues contain lower levels of the receptor binding protein when compared to laboratory animals that have been used in chronic studies of TCDD toxicity. These data also concur with the conclusions of both Neal (Attachment F) and Tschirley (Attachment G) that man is considerably less sensitive to 2,3,7,8 TCDD toxicity than the laboratory animals used in the chronic toxicity studies. Thus, any extrapolation processes should more correctly be based on the premise that man is less sensitive than the laboratory rodents species that have been used in the chronic toxicity studies of 2,3,7,8 TCDD.

COMMENTS ON THE USE OF THE TOXICITY EQUIVALENT FACTOR (TEF) SCHEME:

Attachment D includes a discussion of this issue and lists the results of two recent in vivo animal toxicity studies conducted to test the assumptions inherent in the use of the TEF scheme. Although the concept of using the TEF scheme derived from results of short-term or in vitro assay may seen to be a readily available and attractive interim procedure, two recent studies have reported a lack of correlation between the TEF predicted toxicity and the actual toxicity observed when subchronic animal studies on the mixture are actually conducted. These data indicate that the use of the TEF derived from short term/enzyme assays substantially overpredicts the outcome of subchronic toxicity tests when they are actually conducted on combinations of dioxin/furan components.

It is our hope that these comments and data will be incorporated into subsequent drafts of this document currently being recommended.

COMMENT : There is virtually no scientifically acceptable evidence to suggest that TCDD should be classified as an initiator.

Syntex, Dow.

RESPONSE: It is the practice of the DHS staff to not classify a carcinogen as a promoter or initiator for the purpose of risk assessment but, to treat any carcinogen as though it worked through a non-threshold mechanism unless there is compelling evidence that it only works through a machanism that has a threshold. A number of recent articles support this practice (IARC 1983; Weinstein 1983; Perera 1984).

Members of the DHS staff did find that a number of test systems used to detect genotoxicity and/or interaction with DNA have given negative results when TCDD was tested. However, there are some other test systems or at least some studies with these test systems that have shown that TCDD can cause a mutagenic or a clastogenic effect. There is also evidence that TCDD can be metabolically activated, although there is no evidence to show that these reactive metabolites bind to cellular DNA in vivo. Members of the the DHS staff feel it is inappropriate to discount these positive studies just because they are numerically outnumbered by negative studies.

Our conclusion as stated on page 7-6 was, "Even with the addition of new data, findings are ambiguous or contradictory and

This document contains Department of Health Services (DHS) staff responses to public comments concerning "Health Effects of 2,3,7,8-Tetrachloro-p-dioxin and Related Compounds," which were transmitted to us by the Air Resources Board (ARB) on 6 August 1985. These comments were abstracted from letters sent to ARB by:

- Dow Chemical Company
- Southern California Edison Company (SCE)
- Chemical Manufacturers Association (CMA)
- McKenna, Conner, and Cuneo
- Syntex, Inc.

Since April 1985, when the health effects document was written, ARB staff have revised, slightly upward, their estimates of ambient air exposure. Furthermore, based on the public comments received and re-evaluation of toxicological evidence, DHS staff have produced another exposure scenario, Scenario 4, which is described on pp 10-11 as an addition to the scenarios given in Chapter 10 of the original document. Accordingly, we are also providing revised tables for PCDD and PCDF potencies, and for excess cancer risk estimates, which reflect these changes and additions.

RESPONSE: There are studies that indicate TCDD induces a carcinogenic response through a promoter type mechanism. The mechanism or mechanisms are not understood and infact appear to differ quantitatively and qualitatively from that of a classical promoter 12-0-tetradecanoylphorbol-13-acetate (TPA) as discussed on pages 9-3 and 9-4 of the document. As a promoter, TCDD may have an important interaction with other air pollutants that are known initiators. However, there is little or no information on how the risk from such interactions could be quantitated. In addition, staff members of DHS believe that by quantitating the carcinogenic risks of TCDD based on a non-threshold model, the risks posed to the public through the promoting activity of TCDD will likely not be significant.

COMMENT : The use of the multistage model is inappropriate and produces unreasonably high risk estimates of PCDD and PCDF carcinogenicity.

Sothern California Edison Co. (SCE), Syntex.

RESPONSE: Theoretically, a model which best describes the biological processes would be the model of choice. However, neither cancer induction and promotion, nor detoxification and DNA repair mechanisms are understood well enough to provide an explicit form for a mathematical curve relating dose to cancer risk. Empirically, several different models can be fitted to most data sets.

must be considered inadequate from which to draw a conclusion about TCDD's genotoxicity or clastogenic activity". Because staff members of DHS feel there is uncertainty about TCDD's ability to act as an initiator, the staff cannot conclude that there is compelling evidence that TCDD induces a carcinogenic effect solely through non-genotoxic actions.

COMMENT : The use of a non-threshold based model for risk assessment is inappropriate.

Dow, Syntex.

RESPONSE: This comment is based on the opinion of the commenters that TCDD induces a carcinogenic effect only as a promoter and not as an initiator and therefore, a threshold approach should be used. As previously discussed, staff members of DHS do not classify carcinogens as initiators or promoters for the purpose of risk assessment but, treat any carcinogen as though it worked through a non-threshold mechanism unless there is compelling information to believe otherwise. The staff does not believe there is sufficent information to discount the possibility that TCDD also acts as an initiator.

COMMENT : The impact of both initiator and promoter mechanisms of TCDD carcinogenesis should be reflected in the risk assessment.

McKenna, Conner, and Cueno (McKenna).

COMMENT : Use of the more conservative Weibull model for the risk assessment is more appropriate than using the multistage model.

McKenna.

RESPONSE: With the TCDD data set used in the low-dose expolation of excess cancer risk the Weibull and gamma multi-hit models gave higher risk estimates than did the multistage model. In the computations of excess risk with the Weibull and gamma multi-hit models, power terms are derived for the exposure levels. These terms must be greater than zero but they can be less than one. However, biologically these terms represent the number of hits or interactions between the cell or tissue and the carcinogen needed to induce cancer. Since it is not reasonable to have less than one interaction, when these power terms are less than one the models no longer are biologically plausible. Unfortunately, when the terms are less than one, these models will make the dose-response curve supralinear, that is the estimated excess risk is greater than would be estimated by a straight line to the origin. When the power term in the Weibull model is not allowed to be less than one, the estimated excess risk is similar to that determined by the multistage model (personal communication with Kenny Crump 9/18/85). Therefore, members of the DHS staff feel that results from low dose extrapolations using the multistage model represent best estimate of the maximum excess lifetime cancer risk from exposure to TCDD.

The choice of mathematical models to represent dose-response relationships therefore involves a substantial element of scientific judgement. The considerations for developing or selecting a risk model appropriate for low dose extrapolation include: biologic plausibility, interpretability of the estimated parameters, fit of the model to the observed dose-response relationship, the degree of linearity in the low dose region, and flexibility to take account of survival variation. The relative importance of these considerations depends on the specific data sets available.

The use of mechanistic models, like the multistage and gamma multi-hit models, rather than tolerance distribution models, such as the probit and logistic models, reflects an effort to utilize as fully as possible the current knowledge on carcinogenic processes. From this point of view, the staff of DHS considers the probit and logistic models the least appropriate choices for low dose extrapolation, and the mechanistic models the most biologically plausible.

Because the staff of DHS attempts to provide health-conservative estimates of low dose risk, we frequently rely on the multistage model. This is because 1) it has a plausible biological basis, 2) it has the advantage over other mechanistic models of being essentially linear at low doses, 3) it can fit a variety of empirical data sets reasonably well, and 4) it is more flexible than the one-hit model.

for further risk estimations. This is the same unit risk calculated using data from the Hildebrandt (1983) evaluation which did find two hepatocellular carcinomas in the treated animals.

COMMENT : Variability in spontaneous tumor incidence indicates tumor incidences in Table 8.1-4 and 8.1-5 are not due entirely to TCDD exposure.

Dow.

RESPONSE: Staff members of DHS have reviewed the tumor incidence data of those tumors listed in Tables 8.1-4 and 8.1-5 of the document. There were six individual control groups of 25 animals for each species and sex used in the NTP bioassays on TCDD. Three groups were untreated controls and three groups were vehicle controls. The vehicle control groups were combined for statistical analysis by NTP and the combined data were listed in Tables 8.1-4 and 8.1-5. In a number of cases, when individual untreated or vehicle control groups were compared to the high dose treated group for a particular tumor incidence, the difference was not statistically significant. However, the control group tumor incidence was never greater than that of the treated group. In two cases, subcutaneous tissue fibromas in male rats and histiocytic lymphoma in female mice, did staff members of DHS feel that the tumors may not be a result of TCDD exposure. Therefore, these tumor types will be removed from those listed in the tables.

COMMENT : Use of neoplastic nodule incidence with HexaCDD exposure without actual cancer induction is inappropriate.

Dow.

RESPONSE: The tumor incidence data used to estimate excess lifetime cancer risk from exposure to HexaCDD was that reported by Squire (1983). Squire performed one of four pathological evaluations on liver tissue sections from female rats that were part of the National Toxicology Program (NTP) carcinogenicity bioassays on HexaCDD. Although Squire only reported finding neoplastic nodules, two to four animals in the high dose group were reported to have hepatocellular carcinomas by the other pathologists, as shown in Table 8.1-7 of the document. DHS staff agrees with the International Agency for Research on Cancer (1980) that benign and malignant tumors of the same cell type may be grouped together for statistical analysis when they appear at the same site and it is believed that the benign tumor can progress to a malignant type. Therefore, the staff members of DHS considered HexaCDD to have induced a carcinogenic response since, in all four pathological evaluations there was a statistically significant increase in the tumor incidence. Data from three of the four pathological evaluations were adequate to be used for low-dose risk extrapolation by the multistage model. The results of these extrapolations differed by less than a factor of 2.4, as was shown in Table 10.3-3 of the document. The unit risk obtained from modifying the Squire data was used

It represents a consensus recommendation on science policy. Consequently, assessors and risk managers are urged to use informed discretion when deciding to what situations the procedure can be appropriately applied." The CDWG also states the same concerns about this approach as those expressed by DHS staff members, although they ultimately find that there is sufficient scientific support for the approach. DHS staff members believe a more health-conservative approach is needed because of the uncertainty involved in the EPA approach.

COMMENT: Scenarios 1 and 2 of the risk assessment are based on inappropriate assumptions.

SCE, CMA, Dow.

RESPONSE: The commenters generally state that scenarios 1 and 2 are not valid because they do not take into account all the toxicity information about the different PCDDs and PCDFs. Almost all this information, however, is short-toxicity data or from in vitro studies. As previously stated, DHS staff members do not believe this is adequate to estimate the carcinogenic potency of compounds. The DHS staff made one qualitative assumption for both scenarios that, based on structural similarities, all tetra-, penta-, hexa-, and hepta-chlorinated dioxins and dibenzofurans are potentially carcinogenic. A second qualitative assumption made for scenario 2 limited the potentially carcinogenic PCDDs and PCDFs to those that are chlorinated at the

COMMENT : Scenario 3 of the risk assessment is the best approach to estimating the carcinogenic potency of different polychlorinated dioxin and furan compounds in the absence of carcinogenicity data.

Chemial Manufactures Association (CMA), SCE, Dow.

RESPONSE: Scenario 3 differs from scenarios 1 and 2 of the document in that it assigns more specific potency values to the compounds based both on their degree of chlorination and the ring position of the chlorines. The potency values used were from a report prepared by staff members of the Environmental Protection Agency (EPA) (Bellin and Barnes 1984; personal communications with Barnes 1985). This report has subsequently been drafted as a position document by EPA under the auspices of the Chlorinated Dioxins Work Group (CDWG), although it is not believed that EPA has yet adopted this position document. The potency values were determined from structure-activity relationships based on acute and subacute toxicity and in vitro studies. The commenters generally state that these values represent the best use of the available toxicity data on these compounds. Staff members of DHS, however, believe there is a major problem with this approach because there are not sufficient data to indicate that in vitro and short-term toxicity studies can predict what will occur after long-term exposures. In the EPA position document the CDWG states that "The CDWG acknowledges that this procedure is not based on a thoroughly established scientific foundation.

PCDDs and PCDFs are considered potentially carcinogenic. The carcinogenic potency of TCDD and the HexaCDDs is based on the available experimental evidence. Because of structural similarity between the PCDDs and PCDFs the carcinogenic potencies of TetraCDF and HexaCDFs is assumed to be equivalent to that of the respective dioxin congeners. Although, the observed structure-activity relationship indicates that penta- and heptachlorinated isomers will be less potent than the tetra- and hexachlorinated isomers, respectively, DHS staff members do not believe there is sufficient information to estimate the potency of these compounds other than assuming they are equally potent to the next lowest chlorinated homologue group. Using these potency values the estimates of total carcinogenic potency for a mixture of PCDDs and PCDFs from municipal solid waste incinerator emissions are given in revised Tables B-1 and B-2 of the document (see attachments). The maximum estimated number of excess lifetime cancers, based on scenario 4, that may occur at low and high exposure estimates are 2 and 38 per million persons exposed for a lifetime, repectively. These estimates are in between those obtained using scenarios 2 and 3 as seen in revised Table 10.4-1 (see attachments). At this time the staff of DHS considers scenario 4 is the most appropriate scenario to use for assessment of carcinogenic risk from exposure to mixtures of PCDDs and PCDFs, although scenarios 1 and 2 may also be used.

2,3,7,8 positions and have only 4,5,6, or 7 chlorines. The quantitative assumption made is that for compounds not tested, their carcinogenic potency is equivalent to that of TCDD, which is believed to be the most potent. DHS staff members believed that scenario 2 was the most appropriate, based the on the available information. However, after reevaluation of scenario 2 staff members of DHS have concluded that the scenario fails to fully use the available data. As part of scenario 2, the potency value assigned to the HexaCDDs is based on actual experimental data while the potency values for the other homologue groups are set equal to that of TCDD. Staff members of DHS feel that the HexaCDD toxicity data can be more fully utilized using the following reasoning:

1) Most of the 2,3,7,8-PCDDs and PCDFs are considered potential human carcinogens in scenario 2 because of their similarity in structure to TCDD and 2,3,7,8-HexaCDDs.
2) There is evidence that increased chlorination of the dioxin molecule over the four chlorines on TCDD reduces the carcinogenic potency of the 2,3,7,8-PCDDs.
3) The structural similarity between HexaCDDs and HexaCDFs suggest that they should act in a quantitatively similar fashion.
4) If the structure-activity relationship is correct the HeptaCDDs and -CDFs will be less potent than the hexachlorinated compounds.

Using this line of reasoning, a forth scenario is proposed. As in scenario 2, only tetra-, penta-, hexa-, and heptachlorinated

RESPONSE: There is a discussion on the interaction of TCDD and other carcinogens presented in chapter 9 of the document. As described in this chapter TCDD has been found to act as a promoter and cocarcinogen but also has been found to inhibit carcinogenicity in some experimental systems. Synergism or other interactions between TCDD and non-carcinogens are not well studied. Because of the complexities of such interactions, staff members of DHS do not feel this issue can be discussed in depth based on current knowledge and that such a discussion could not impact the risk assessment because of the uncertainties involved.

COMMENT: Issue of decreased bioavailability of dioxins adsorbed to soil or dust particles is not adequately discussed and factored into the risk assessment.

Dow.

RESPONSE: Bioavailability of PCDDs was discussed in chapter 4 and how it could be factored into the risk assessment was discussed in Appendix A. The conclusion was that it is not necessary to include such factors because they did not make a large difference in the risk assessment, under the assumptions used, to qualitatively out weigh the uncertainties involved with these factors. Further evaluation of this point has indicated that bioavailability factors could make a much larger difference than first realized. However, the uncertainty is still great enough,

COMMENT : The conclusion that all PCDD and PCDF compounds chlorinated in the 2,3,7,8-positions, especially HeptaCDD and -CDF, are carcinogenic is not generally accepted.

CMA, Dow, Syntex.

RESPONSE: Because only 2,3,7,8-TCDD and a mixture of two 2,3,7,8-HexaCDD isomers have been assayed for carcinogenicity, these are the only compounds staff members of DHS consider to have been shown to be carcinogenic to animals and therefore, potentially carcinogenic to humans. As previously stated, there have been no other long-term studies on PCDDs and PCDFs that contain seven chlorines. Since the tested compounds were found to be so potent, and the congeners are structurally similar, staff members of DHS have concluded that the other congeners are potentially carcinogenic and that this potential needed to be addressed. DHS is not alone in this position. In fact, the CDWG, which many commenters indicated used valid methods to estimate the potency of the various homologue groups, included the same PCDDs and PCDFs that staff members of DHS considered in their assessment of carcinogenic risk.

COMMENT: Synergism between TCDD and other carcinogens and non-carcinogens should be discussed.

McKenna.

as statistically significant. A few cases of soft-tissue sarcomas have occurred among American workers with potential dioxin exposure. But whether these represent a greater than expected number is still being debated.

The limitations of these data that make them inadequate to demonstrate carcinogenicity or non-carcinogenicity include:

1) Uncertain exposure to TCDD--usually exposures occurred in the past when there were no sensitive measures of exposure levels. "Exposure" in these studies is typically based on job title, self-reported use of chemical products which may have contained TCDD as a contaminant, or exposure to a chemical process thought to have liberated TCDD.

2) Exposure for a short time--many of the occupationally exposed subjects were exposed only briefly (e.g., during an accidental release), or worked in a possibly contaminated environment for a short time.

Negative epidemiologic studies with small sample sizes, low power, and uncertain exposures are insufficient to demonstrate non-carcinogenicity. Therefore, in view of the conflicting results, DHS staff concur with IARC that the evidence of carcinogenicity to humans is inadequate. But based on the available human and animal data, DHS staff believe that TCDD should be considered as a potential human carcinogen.

that the use of equal bioavailability should be used in the risk assessment.

COMMENT: Results from epidemiologic studies show that man is not at increased risk of cancer due to TCDD exposure.

Syntex, Dow.

RESPONSE: Results from some epidemiologic studies suggest that dioxins may cause cancer in humans. Other studies have shown no effect. Based on these results, DHS staff must conlude that the available human data are insufficient to declare whether TCDD is or is not carcinogenic to man.

Three case/control studies in Sweden showed that patients with soft-tissue sarcomas and nasopharyngeal cancer had reported a significantly greater than expected frequency of exposure to chlorinated phenols and phenoxyacetic acids that were considered to have contained TCDD as a contaminant. The findings of the Swedish case/control studies were not repeated in a similar study conducted in New Zealand.

Longitudinal studies of occupational exposure to dioxin show small or no increases in cancer. A few studies reported increases in cancer mortality on the order of 20% to 80%. However, these studies were too small to detect such increases

Revised Table B-1
Estimates of Total Carcinogenic Potency (Relative to 2,3,7,8-TCDD)
for a Mixture of PCDDs from Municipal Solid Waste Incinerator Emissions

Homologue	Proportion of Homologue in Emissions*	Proportion of Isomer in Homologue Group**	Proportion of Isomer in Emissions	Scenario 2+ Potency Score	Scenario 2+ Equivalent TCDD Proportion	Scenario 3++ Potency Score	Scenario 3++ Equivalent TCDD Proportion	Scenario 4+++ Potency Score	Scenario 4+++ Equivalent TCDD Proportion
TetraCDD	0.09	2,3,7,8 isomer = 0.045	0.004	1.00	0.004	1.00000	0.0041	1.00	0.004
		Non 2,3,7,8 = 0.955	0.086	0.00	0.000	0.01000	0.0009	0.00	0.000
PentaCDD	0.12	2,3,7,8 isomer = 0.071	0.009	1.00	0.009	0.20000	0.0017	1.00	0.009
		Non 2,3,7,8 = 0.929	0.112	0.00	0.000	0.00200	0.0002	0.00	0.000
HexaCDD	0.20	2,3,7,8 isomer = 0.300	0.060	0.03	0.002	0.03000	0.0016	0.03	0.002
		Non 2,3,7,9 = 0.700	0.140	0.00	0.000	0.00040	0.0001	0.00	0.000
HeptaCDD	0.47	2,3,7,8 isomer = 0.500	0.235	1.00	0.235	0.00100	0.0002	0.03	0.007
		Non 2,3,7,8 = 0.500	0.235	0.00	0.000	0.00001	0.0000	0.00	0.000
OctaCDD	0.11	2,3,7,8 isomer = 1.000	0.110	0.00	0.000	0.00000	0.0000	0.00	0.000
	1.0		1.0	Total =	0.25	Total =	0.01	Total =	0.022

* From Tiernan, et al. (1983)
** Assumes equal distribution of isomers in homologue group (cf. Olie, et al., 1982)
\+ Scenario 2: Relative potency is one for isomers chlorinated on 2,3,7,8 position, 0 otherwise, 0 for OctaCDD, and 0.03 for HexaCDD (from multistage model)
++ Scenario 3: Relative potencies from Bellin and Barnes (1984), and Barnes (personal communication, 1985)
+++ Scenario 4: Relative potency for 2,3,7,8-chlorinated isomers of PentaCDD is equal to 2,3,7,8-TCDD, relative potency for 2,3,7,8-chlorinated isomers of HeptaCDD is equal to 2,3,7,8 isomer HexaCDD.

References

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Revised Table 10.4-1

Risk Estimates for Total PCDD/PCDF
Under Different Exposure Assumptions

	High Exposure Estimates+		Low Exposure Estimates+	
	PCDDs: 1.3×10^{-2} ng/m^3		PCDDs: 0.08×10^{-2} ng/m^3	
	PCDFs: 2.7×10^{-2} ng/m^3		PCDFs: 0.17×10^{-2} ng/m^3	
Potency Scenarios for a Mixture of PCDD/PCDF	Equivalent TCDD Dose ($\times 10^{-2}$ ng/m^3)	Upper 95% Estimated Excess Lifetime Cancers (per million)	Equivalent TCDD Dose ($\times 10^{-2}$ ng/m^3)	Upper 95% Estimated Excess Lifetime Cancers (per million)
Scenario 1*	4.0	1500	0.25	95
Scenario 2**	0.87	330	0.054	20
Scenario 3***	0.02	8	0.001	< 1
Scenario 4****	0.10	38	0.006	2

\+ High and low exposure estimates revised to reflect new ARR estimates

* Assumes all PCDD is 2,3,7,8-TCDD, or that all PCDD/PCDF is equipotent to 2,3,7,8-TCDD.

** Uses potencies derived from bioassays for 2,3,7,8 chlorinated TCDD and HexaCDD; assumes all other isomers chlorinated on 2,3,7,8 positions are equipotent to 2,3,7,8-TCDD; assumes that isomers not chlorinated on the 2,3,7,8 positions are non-carcinogenic; assumes that OctaCDD/CDF are noncarcinogenic.

*** Uses potencies derived from bioassays for 2,3,7,8 chlorinated TCDD and HexaCDD; for other isomers, uses potency assumptions from Bellin and Barnes (1984), and Barnes (personal communication, 1985).

**** Uses potencies derived from bioassays for 2,3,7,8 chlorinated TCDD and HexaCDD; assumes assumes that 2,3,7,8 chlorinated isomers of TetraCDF, PentaCDD, and PentaCDF are equipotent to 2,3,7,8-TCDD; assumes that 2,3,7,8 chlorinated isomers of HexaCDF, HeptaCDD, and HeptaCDF are equipotent to 2,3,7,8 isomer HexaCDD; assumes that isomers not chlorinated on the 2,3,7,8 positions are non-carcinogenic; assumes that OctaCDD/CDF are noncarcinogenic.

Revised Table B-2
Estimates of Total Carcinogenic Potency (Relative to 2,3,7,8-TCDD)
for a Mixture of PCDFs from Municipal Solid Waste Incinerator Emissions

Homologue	Proportion of Homologue in Emissions*	Proportion of Isomer in Homologue Group**	Proportion of Isomer in Emissions	Scenario 2+ Potency Score	Scenario 2+ Equivalent TCDD Potency	Scenario 3++ Potency Score	Scenario 3++ Equivalent TCDD Potency	Scenario 4+++ Potency Score	Scenario 4+++ Equivalent TCDD Potency
TetraCDF	0.31	2,3,7,8 isomer = 0.026	0.008	1.00	0.008	0.10000	0.000800	1.00	0.008
		Non 2,3,7,8 = 0.974	0.302	0.00	0.000	0.00100	0.000302	0.00	0.000
PentaCDF	0.19	2,3,7,8 isomer = 0.072	0.0137	1.00	0.0137	0.10000	0.001370	1.00	0.0137
		Non 2,3,7,8 = 0.928	0.176	0.00	0.000	0.00100	0.000176	0.00	0.000
HexaCDF	0.21	2,3,7,8 isomer = 0.252	0.053	1.00	0.053	0.01000	0.000530	0.03	0.0016
		Non 2,3,7,8 = 0.748	0.157	0.00	0.000	0.00010	0.000016	0.00	0.000
HeptaCDF	0.26	2,3,7,8 isomer = 0.500	0.130	1.00	0.130	0.00100	0.000130	0.03	0.0039
		Non 2,3,7,8 = 0.500	0.130	0.00	0.000	0.00001	0.000001	0.00	0.000
OctaCDF	0.02	2,3,7,8 isomer = 1.000	0.020	0.00	0.000	0.00000	0.000000	0.00	0.000
	1.0		1.0	Total = 0.20		Total = 0.003		Total = 0.027	

* From Tiernan, et al. (1983)
** Assumes equal distribution of isomers in homologue group (cf. Olie, et al., 1982)
\+ Scenario 2: Relative potency is 1 for isomers chlorinated on 2,3,7,8 position, 0 otherwise, and 0 for OctaCDF
++ Scenario 3: Relative potencies from Bellin and Barnes (1984), and Barnes (personal communication, 1985)
+++ Scenario 4: Relative potencies for 2,3,7,8-chlorinated isomers of Tetra- and PentaCDF are equal to 2,3,7,8-TCDD, relative potencies for 2,3,7,8-chlorinated isomers of Hexa- and HeptaCDF are equal to 2,3,7,8 isomer HexaCDD.

APPENDIX D

WORLDWIDE DETECTION OF CDDs AND CDFs

APPENDIX D

WORLDWIDE DETECTION OF CDDs AND CDFs

Over the past 10 years, extensive data have been collected on both sources and levels of CDDs and CDFs in the environment. While 2,3,7,8-tetraCDD was the initial congener of concern and most of the earlier results report only this compound, the realization that other congeners chlorinated at the 2,3,7, and 8 positions also possess significant toxicity has lead to greater efforts to search for them in a variety of environmental compartments.

This appendix presents an overview of significant CDD and CDF contamination incidents worldwide, as well as a limited number of examples where these compounds were detected in California.

ENVIRONMENTAL CONTAMINATION--WORLDWIDE

This section summarizes reported worldwide incidents, excluding California, where releases of CDDs and CDFs into the environment have occurred. (California data are discussed separately in this appendix). Ranges of representative worldwide values of CDD and CDF isomer groups or individual congeners are presented in four separate tables.

Table D.1 shows concentrations of the CDD and CDF isomer groups found in a variety of commercial products. Table D.2 provides ranges of concentrations reported in water, soil, sediment and air. Table D.3 presents reported values found in biota (fish, animals, plants) and Table D.4 lists reported ranges of specific congeners detected in humans (adipose, liver, blood, milk). For each table there is a discussion of the major incidents which led to the CDD or CDF exposure.

Products Containing CDDs and CDFs--Table D.1

The CDDs and CDFs are not intentionally produced, but are found as impurities in a variety of commercial products such as wood preservatives (chlorinated phenols), phenoxy herbicides, PCBs and diphenyl ether herbicides. The amount of these impurities depends upon the method of preparation. U.S. EPA (1978), Da Roos et al., (1981), Rappe (1984) and Buser et al. (1976) have reported the results of some typical analyses of commercial products containing CDDs and CDFs.

Firestone (1978) and U.S. EPA (Esposito et al., 1980) have found tetraCDD in the ppt range in drums of 2,4,5-trichlorophenoxy acetic acid (2,4,5-T, a component of Agent Orange). Earlier, in the 1960's, the mean level of 2,3,7,8-tetraCDD in Agent Orange preparations was 1.98 ppm (U.S. EPA, 1985b). At the present time, producers claim that their products contain less than 0.1 ppt of 2,3,7,8-tetraCDD (U.S. EPA, 1985b).

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TABLE D.1 (continued)

CDD AND CDF CONCENTRATIONS (ppm) IN COMMERCIAL PRODUCTS

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Unpurified Commercial PCP-Na Pellets (Buser and Bosshardt, 1976)	0.08	0.03	0.25	2.8	5.1	0.02	0.13	4.1	13	8.6
Unpurified Commercial PCP-Na Granules (Buser and Bosshardt, 1976)	0.05	<0.03	3.4	40	115	<0.02	0.05	11	50	24
Phenoxy Herbicides										
2,4,5-T (acids, esters, & formulated products) (2,3,7,8-specific) (U.S. EPA, 1985b)	0.01-0.08	NA	NA	NA	NA	NA	NA	NA	NA	NA
Agent Orange (mixture of 2,4,5-T & 2,4-D) mean value (Firestone, 1978; Esposito et al., 1980)	.002-.054	NA	NA	NA	NA	NA	NA	NA	NA	NA
Commercial PCB										
Aroclor 1248 (Rappe, 1984)	NA	NA	NA	NA	NA	0.5	1.2	0.3	NA	NA
Clophen A-60 (Rappe, 1984)	NA	NA	NA	NA	NA	1.4	5.0	2.2	NA	NA
Phenoclor DP-6 (Rappe, 1984)	NA	NA	NA	NA	NA	0.7	10.0	2.9	NA	NA

APPENDIX D

TABLE D.1

CDD AND CDF CONCENTRATIONS (ppm) IN COMMERCIAL PRODUCTS

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Wood Preservatives										
Wood Treatment Oil containing 4.5% PCP (2,3,7,8-specific) (Da Roos et al., 1981)	0.001	0.033	0.574	0.256	3.99	0.018	0.137	1.813	0.114	0.711
Unpurified Commercial PCP (Rappe, 1984)	<0.1	<0.1	2.5	175	500	<0.1	<0.1	<0.3	19	25
Unpurified Commercial PCP (88% Penta) (U.S. EPA, 1978)	NA	NA	4	125	2500	NA	NA	30	80	80
Technical Grade PCP Reduced by Distillation (89% Penta) (U.S. EPA, 1978)	NA	NA	1.0	6.5	15	NA	NA	1.0	1.8	1.0
Unpurified Commercial PCP flakes (Buser and Bosshardt, 1976)	<0.02	<0.03	5.2	95	280	0.02	0.40	28	200	230
Unpurified Commercial PCP-Na Powder (Buser and Bosshardt, 1976)	0.16	0.03	<0.03	0.3	1.2	<0.02	<0.03	0.20	1.2	3.0

Rappe (1984) examined a series of commercial PCBs of both United States and European manufacture. He reported that the most abundant CDFs had the same retention time as 2,3,7,8-tetraCDF and 2,3,4,7,8-pentaCDF.

Yamagishi et al. (1981) found levels of both CDDs and CDFs in preparations of the commercial diphenyl ether herbicides CNP, NIP, and X-52. The major tetraCDDs identified were 1,3,6,8 and 1,3,7,9 isomers. The 2,3,7,8-tetraCDD isomer was not found in these samples.

CDDs and CDFs in Water, Soil, Sediment and Air--Table D.2

Thibodeaux (1983) assessed quantities of 2,3,7,8-tetraCDD in surface water, soil, creek and pond sediment resulting from an herbicide production facility in Jacksonville, Arkansas that practiced onsite disposal. The waste was stored in metal drums buried onsite or deposited on soil or into water bodies within the plant boundary. The plant had been manufacturing 2,4-D and 2,4,5-T since 1958. 2,3,7,8-TetraCDD was not detected until spring 1979, after which approximately 1000 soil, air, water, and sediment samples were taken by government and company representatives. The pond sediment from onsite appeared to be the most contaminated source of 2,3,7,8-tetraCDD (22.1 $\pm$ 2.1 ppb 2,3,7,8-tetraCDD).

Pereira et al. (1985) performed geochemical investigations of pond sludge, groundwater and porous media from the unsaturated and saturated zones at a wood treatment facility in Pensacola, Florida in July 1983. The facility had been discharging creosote and PCP into two unlined surface impoundments on site, resulting in contamination of the underlying sand and gravel aquifer. It had operated from 1902 to 1981 but has since discontinued all operations. Researchers found that CDDs had migrated both vertically and horizontally in the subsurface and were present at considerable distances from the source of contamination. Ground water samples taken at various depths were generally in the ppt range (wet weight) whereas concentrations of CDDs in the porous media of the saturated and unsaturated zones were in the ppb range (dry weight). Significant concentrations of hexaCDD, heptaCDD, and octaCDD associated with the sediment and bottom material of two surface impoundments were reported in the ppm range (wet weight).

In Missouri, horse arenas were sprayed in 1971 with 2,000 gallons of a dust control solution made from trichlorophenol (triCP) distillation products mixed with motor oil. Subsequently, animals living on or near the arenas died and several children became ill. Sampling of the soil in the arenas between 1971 and 1972 indicated very high (ppm) concentrations of tetraCDD, triCP, and PCBs. After the soil was excavated twice from one arena in 1974, no detectable concentrations of tetraCDD or PCB and only trace amounts of triCP were found (Reggiani, 1980). Eight years

TABLE D.1 (continued)

CDD AND CDF CONCENTRATIONS (ppm) IN COMMERCIAL PRODUCTS

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Diphenyl Ether Herbicides										
CNP (Yamagashi et al., 1981)	14.0	37	0.8	NA	NA	0.4	1.0	0.2	NA	NA
NIP (Yamagashi et al., 1981)	0.38	0.05	ND	NA	NA	0.29	ND	ND	NA	NA
X-52 (Yamagashi et al., 1981)	0.03	0.01	ND	NA	NA	0.32	0.08	ND	NA	NA

ND - Not Detected
NA - Not Available

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Soil										
Surface soil (upper 7 cm) from Seveso, Italy after industrial accident. Soil density 1.4 kg/liter (Reggiani, 1980)										
Highest value closest to factory	55	NA	NA	NA	NA	NA	NA	NA	NA	NA
Highest value in formerly inhabited area	20	NA	NA	NA	NA	NA	NA	NA	NA	NA
Limit for evacuation	0.1	NA	NA	NA	NA	NA	NA	NA	NA	NA
Missouri horse arenas that were sprayed for dust control using TCDD contaminated industrial waste residues (Reggiani, 1980)										
Moscow Mills Arena in Lincoln County, August, 1971	31,800-33,000	NA	NA	NA	NA	NA	NA	NA	NA	NA
New Bloomfield Arena in Phelps County,	220-850	NA	NA	NA	NA	NA	NA	NA	NA	NA
St. James Arena in Calloway County, August, 1974	120	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description		Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Wastewater											
Surface Water at a 2,4-D & 2,4,5-T production facility in Jacksonville, Arkansas (2,3,7,8-Specific) (Thibodeaux, 1983)		0.014	NA	NA	NA	NA	NA	NA	NA	NA	NA
Waste water from a wood preservation plant in Ottawa, Canada (Lao et al., 1983)		0.0012	0.0083	0.034	0.122	0.258	0.0007	0.0015	0.0115	0.048	0.038
Rainwater runoff sump sludge at a wood preservation plant (site unidentified) (U.S. EPA, 1986f)		ND	ND	<4.5	111	262	ND	ND	ND	<2.3	23.6
Ambient Water											
Perched ground water (oil sheen filtered) (site unidentified) (U.S. EPA, 1986f)		ND	ND	0.31	5.8	50	ND	ND	722	6000	23000
	Ft.										
Ground water from	20	NA	NA	0.061	1.500	3.900	NA	NA	NA	NA	NA
an abandoned wood	40	NA	NA	0.0019	0.0046	0.0217	NA	NA	NA	NA	NA
treatment facility	60	NA	NA	0.021	0.034	0.039	NA	NA	NA	NA	NA
in Pensacola,	80	NA	NA	ND	ND	ND	NA	NA	NA	NA	NA
Florida - depth to	100	NA	NA	ND	0.0004	0.0014	NA	NA	NA	NA	NA
water in feet. (Pereira et al., 1985)											

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Sediment										
From Swiss Lakes near municipal incinerators (dry weight) (average of 3 labs)(Czuczwa et al., 1985)	ND	.05	.13	.35	1.3	.08	ND	.02	.20	.15
Samples from a refuse dump near Amsterdam, Holland (dry weight) 20% organic content (Heida, 1983)										
- within dump area (range)	.844-5.062	NA	NA	NA	NA	NA	NA	NA	NA	NA
- outside dump area (range)	.055-.611	NA	NA	NA	NA	NA	NA	NA	NA	NA
From a wood treatment facility in Pensacola, Florida (average of 2 samples) (Pereira et al., 1985)	NA	NA	373	10,750	94,000	NA	NA	NA	NA	NA
Sludge										
Pond sludge from a wood treatment facility in Pensacola, Florida (average of 2 samples) (Pereira et al., 1985)	NA	NA	365	9,020	39,500	NA	NA	NA	NA	
Dust										
Samples from Dow Chemical Research Building in Midlands, Michigan (Esposito et al., 1980)	.5-2.3	NA	9-35	140-1200	650-7500	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Soil										
Surface soil from on-site disposal of 2,4-D and 2,4,5-T in Jacksonville, Arkansas (2,3,7,8-specific) 5 samples (Thibodeaux, 1983)										
Average	1.3	NA	NA	NA	NA	NA	NA	NA	NA	NA
Range	ND to 2.9	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface soil from Times Beach, Missouri. Samples taken in early December 1982 highest concentration detected of 2,3,7,8-tetraCDD) (Kleopfer, 1985)	1200	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment										
On-site disposal of 2,4,D and 2,4,5-T in Jacksonville, Arkansas (2,3,7,8-Specific) (Thibodeaux, 1983)										
Creek Sediment (average of 5 samples)	.77	NA	NA	NA	NA	NA	NA	NA	NA	NA
Range	ND to 1.8	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pond Sediment (average of 3 samples)	22.1	NA	NA	NA	NA	NA	NA	NA	NA	NA
Range	±2.1	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Air										
Airborne particulates from municipal incinerator sample over a 5-hour period during which 6.8 m^3 of stack gas was released at the Hampstead Refuse Recovery Corporation, Nassau County, New York (Tiernan et al., 1983)	.38	.53	.85	2.00	.49	2.60	1.60	1.80	2.20	.17
Airborne particulates from Hamilton Municipal Incinerator, Ontario, Canada, 1983-1984 (average of 3 tests) (NRCC, 1984)	1.70	1.08	.48	.33	.12	5.64	3.03	.47	.15	.05

NA = Not available
ND = Not detected (values in parentheses are limits of detection)

APPENDIX D

TABLE D.2 (continued)

CDD AND CDF CONCENTRATIONS (ppb) IN WATER, SOIL, SEDIMENT, AND AIR

Description	Tetra CDD	Penta CDD	Hexa CDD	Hepta CDD	Octa CDD	Tetra CDF	Penta CDF	Hexa CDF	Hepta CDF	Octa CDF
Soot										
From Binghamton, State Office Building. Each Lab received samples collected at different times and locations (Schecter et al., 1985b)										
Lab 1	<3,000	<2,000	<3,000	7,000	5,000	$1.92_6 \times 10^6$	1.2×10^6	$1.16_6 \times 10^6$	$4.05_5 \times 10^5$	66,000
Lab 2	1,200	5,000	5,000	7,000	2,000	28,000	6.7×10^5	$9.65_5 \times 10^5$	4.6×10^5	40,000
Air										
Fly Ash from a municipal incinerator in Switzerland (NRCC, 1984)	NA	NA	NA	NA	NA	1.0	4.0	30.0	40.0	10.0
Atmospheric dust from Seveso, Italy after industrial accident (2,3,7,8-Specific) (U.S. EPA, 1985b)	.06-.50	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air particulate sample from a waste disposal site near Jacksonville, Arkansas (average concentration of tetraCDD) (U.S. EPA, 1985b)	1.1	NA	NA	NA	NA	NA	NA	NA	NA	NA

CDDs and CDFs were found in annually laminated sediment from Lakes Zurich, Baldegg, and Lugano in Switzerland. OctaCDD predominated, averaging approximately 1.3 ppb. The congener distribution indicated that combustion was the source of CDDs and CDFs in these sediments (Czuczwa et al., 1985).

Combustion is now generally recognized as an important potential source of CDDs and CDFs in the environment. Efforts to determine concentrations in the air have been focused mainly on municipal solid waste incinerators and power plants from other states and Canada (NRCC, 1984; U.S. EPA, 1985b; Tiernan, 1983). The concentration levels in air depend on a host of features including feedstock burned, the facility design and operational variables. Only a few measurements of possible CDD and CDF precursor compounds in incinerator effluents have been made.

Reported CDD and CDF Concentrations in Biota--Table D.3

Kaczmar (1983) examined various species of fish in selected Michigan water systems for residues of 2,3,7,8-tetraCDD. Detectable residues of 2,3,7,8-tetraCDD ranged from 17 to 586 ppt. The significance to this particular study was that the investigator found residues in fish collected upstream of a chlorophenol manufacturing facility. The study suggests that low-level contamination (in the ppt range) of bottom feeding fish is relatively widespread in the industrialized portions of Michigan.

New York State Health Department performed congener specific determination of 2,3,7,8-tetraCDD levels in Great Lakes fish (NRCC, 1981). Of the 76 samples, 2,3,7,8-tetraCDD levels ranged from non-detectable to 162 ppt (detection limit not given). Fish sampled included small mouth bass, lake trout, white sucker, brown bullhead, rainbow trout, coho and chinook salmon, and brown trout. Lakes sampled included Lake Ontario, Lake Erie, Lake Huron, Lake Michigan, and Lake Superior. As a result of this study, New York State issued a health advisory fishing guideline that is more stringent than the FDA's or Canada's. See Chapter 7: Criteria and Standards.)

O'Keefe et al. (1984) examined whole body samples of striped bass from the lower Hudson River in New York for 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF. Concentrations of 2,3,7,8-tetraCDD ranged from 16 to 120 ppt. Striped bass from other locations (Rhode Island coastal waters and Chesapeake Bay, Maryland) had less than 5 ppt. Concentrations of 2,3,7,8-tetraCDF found in striped bass from all three locations ranged from 6 ppt in Chesapeake Bay to 78 ppt in the Hudson River.

later the horse arenas were reinvestigated (Kleopfer, 1985). As of August 1982, 10,734 Missouri soil samples had been processed for analysis of 2,3,7,8-tetraCDD and 92 percent of the results of these analyses had been validated through a quality assurance plan. Of the validated results, 23 percent were positive and above 1.0 ppb, while 2.2 percent were above 100 ppb (identity of excavated vs. non-excavated sites was not given).

In 1981 a fire involving an electrical transformer containing a mixture of PCBs and chlorinated benzenes occurred at a Binghamton, New York office building. This led to contamination of the building structure with CDF and CDD laden soot. Various samples revealed very high concentrations of the CDDs (ppm range) and CDFs (parts per thousand range) (Schecter et al., 1985b). Two years after several cleanup operations, air samples still showed measurable values for selected isomer groups.

Heida (1983) examined tetraCDD in sediment from ponds and canals located near a refuse dump in Amsterdam, Holland. The 2,3,7,8-tetraCDD concentrations originated from 2,4,5-T production. The results showed that the highest concentration of 2,3,7,8-tetraCDD (5.0 ppb) occurred in the main drainage canal near the dump and rapidly decreased outside the dump area. Analysis of eel flesh revealed only two samples (total samples not given) from the sampling site inside the dump area which contained small quantities of 2,3,7,8-tetraCDD (3.9 ppb in body fat).

In 1972, and again in 1973, the streets of Times Beach, Missouri were oiled by a firm specializing in waste oil reclamation. Mixed with the waste oil were impurities ("still-bottoms") from the production of 2,4,5-trichlorophenol (Kleopfer, 1985). The soil was sampled by U.S. EPA in early December 1982 (Kleopfer, 1985), and the highest level of 2,3,7,8-tetraCDD detected was 1,200 ppb in surface soil. The Centers for Disease Control concluded in a risk analysis that levels of 1 ppb or greater of 2,3,7,8-tetraCDD in residential soil represented an unreasonable risk. Subsequently, the town was evacuated and eventually bought out by the government.

One of the most well known incidents of 2,3,7,8-tetraCDD contamination occurred in Seveso, Italy in 1976. Here a plant manufacturing hexachlorophene exploded and contaminated about 700 acres adjoining the plant. Because of the high concentrations of 2,3,7,8-tetraCDD found in the soil, the Italian authorities evacuated 736 people from the area. Up to 5,000 people were believed to have been exposed from the explosion. Monitoring of Seveso soil one year after the accident showed that the highest concentrations of 2,3,7,8-tetraCDD were not present in the topmost soil layer (0.5 cm), but very often in the second (0.5-1.0 cm) or third (1.0-1.5 cm) layers (Reggiani, 1980).

TABLE D.3 (continued)

2,3,7,8-TETRACDD AND TETRACDF CONCENTRATIONS (ppt) IN BIOTA

Description	2,3,7,8-tetraCDD	2,3,7,8-tetraCDF
White perch (whole body) from Cape Vincent in Lake Ontario (range of 4 samples) (NRCC, 1981)	4.9-36	NA
Striped bass (whole body) from Rhode Island coastal waters (range from 5 samples) (O'Keefe et al., 1984)	2.0-5.0	17-50
Striped bass (whole body) from Little Neck Bay, Long Island, New York (range from 4 samples) (O'Keefe et al., 1984)	ND (0.3-7.1) - 39	12-22
Striped bass (whole body) from Newark Bay, New Jersey (range from 4 samples) (O'Keefe et al., 1984)	16-67	20-34
Striped bass (whole body) from Tappen Zee Bridge, Hudson River, New York (range from 4 samples) (O'Keefe et al., 1984)	ND (0.2-2.0) - 33	16-72
Striped bass (whole body) from Poughkeepsie, Hudson River, New York (2 samples) (O'Keefe et al., 1984)	120	74-78
Striped bass (whole body) Chesapeake Bay, Maryland (1 sample) (O'Keefe et al., 1984)	ND (3.5)	ND (13)

APPENDIX D

TABLE D.3

2,3,7,8-TETRACDD AND TETRACDF CONCENTRATIONS (ppt) IN BIOTA

Description	2,3,7,8-tetraCDD	2,3,7,8-tetraCDF
Fish		
Carp (whole body) from Tittabawassee, below dam (1 sample) (Kaczmar, 1983)	17	NA
Carp (whole body) from St. Clair River at Decker's Landing (Lake Huron) (1 sample) (Kaczmar, 1983)	586	NA
Coho salmon (whole body) from Salmon River near Lake Ontario (1 sample) (NRCC, 1981)	31	NA
Coho salmon (whole body) from Spring Brook Weir near Lake Ontario (range of 3 composite samples) (NRCC, 1981)	19-22	NA
Coho salmon (whole body) from Belle Isle on Lake Erie (range from 3 samples) (NRCC, 1981)	0.9-1.4	NA
Coho salmon (whole body) from St. Joseph on Lake Michigan (5 samples) (NRCC, 1981)	ND (2.0-4.9)	NA
Brown trout (whole body) from St. Catherines/Niagara in Lake Ontario (1 sample) (NRCC, 1981)	162	NA
Brown trout (whole body) from Nine Mile Point in Lake Ontario (1 sample) (NRCC, 1981)	8	NA

Several investigators (Esposito et al., 1980) have studied the levels of tetraCDD in wild animals in the contaminated area near Seveso, Italy. Field mice contained tetraCDD concentrations ranging from 70 to 49,000 ppt (mean value 4500 ppt). These mice lived on soil where the upper 7 centimeters varied from 10 to 12,000 ppt of tetraCDD (mean value 3500 ppt). Several rabbits and one snake showed tetraCDD in the liver. Liver samples from domestic birds were analyzed for tetraCDD with negative results.

CDD and CDF Concentrations in Human Tissues--Table D.4

Schecter et al. (1985b) examined human adipose tissue for tetra through octaCDD substituted congeners. All persons examined resided in upstate New York during 1983 to 1984. Tissues from exposed versus unexposed individuals were evaluated. The difference in CDD congener concentrations between the person exposed to the soot from the Binghamton State Office building transformer fire and the control group was surprisingly small for most congeners. Penta- and hexaCDFs were also found in the exposed and, to a lesser extent, in the control population. The high background contamination (in the control group) was believed by the authors to be caused by exposure to technical grade PCP or food containing PCP.

Nygren et al. (1985) performed a study to determine if there was any difference between cancer patients previously exposed to chlorinated phenoxyacetic acids over a ten year period and to unexposed controls. Adipose tissue was excised from each group. There was no difference in levels and pattern between the cancer patients and controls except for the 2,3,4,7,8-pentaCDF congener, which could not be associated with the specific exposure. The congener profile for the mean and individual values were all identical. According to the authors, the data strongly suggests that there is a background concentration of CDDs and CDFs in the general population.

The adipose tissue of Swedish workers exposed to chlorinated phenoxyacetic acids was analyzed for CDDs and CDFs and compared to adipose tissues of unexposed workers (Hardell et al., 1985). Mean levels and ranges are presented in Table D.4. Regarding CDDs, the only significant finding was hexaCDD at levels higher in exposed than in unexposed individuals. The difference was attributed to the 1,2,3,6,7,8-hexaCDD congener. Mean levels of pentaCDF and hexaCDF were significantly higher in exposed versus the unexposed individuals. No difference was found in exposed and unexposed individuals for tetraCDD.

In 1968, over 1500 persons in southwest Japan were exposed by consuming a commercial rice oil accidently contaminated by PCB, CDF and polychlorinated quaterphenyls. In 1979, a similar episode was reported in Taiwan where over 2,000 persons were exposed. These are referred to as the "Yusho" episodes.

TABLE D.3 (continued)

2,3,7,8-TETRACDD AND TETRACDF CONCENTRATIONS (ppt) IN BIOTA

Description	2,3,7,8-tetraCDD	2,3,7,8-tetraCDF
Terrestrial Biota		
Field mouse (whole body) from Seveso, Italy (range of 14 samples) (Esposito et al., 1980)	70-49,000	NA
Hare (liver) from Seveso, Italy (1 sample) (Esposito et al., 1980)	7,700	NA
Toad (whole body) from Seveso, Italy (1 sample) (Esposito et al., 1980)	200	NA
Snake (liver) from Seveso, Italy (1 sample) (Esposito et al., 1980)	2,700	NA
Snake (adipose tissue) from Seveso, Italy (1 sample) (Esposito et al., 1980)	16,000	NA
Earthworm (whole body) from Seveso, Italy (average of 2 samples) (Esposito et al., 1980)	12,000	NA
Cow (milk) from Seveso, Italy (average of 9 samples) (Esposito et al., 1980)	2,196	NA

Footnotes

NA - Not Available
ND - Not Detected
Numbers in parentheses are limits of detection

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

Description		2,3,7,8-TetraCDD	Tetra CDD	1,2,3,7,8-PentaCDD	Penta CDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	Hexa CDD	1,2,3,4,6,7,8-HeptaCDD	Hepta CDD	Octa CDD	Total CDD
7 Swedish workers exposed to phenoxy acids (Hardell et al., 1985)	mean	NA	2.8	NA	11	NA	NA	31	NA	133	443	642
	range	NA	ND-9.0	NA	ND-24	NA	NA	16-68	NA	28-380	154-623	230-914
18 unexposed workers (Hardell et al., 1985)	mean	NA	2.7	NA	8.7	NA	NA	16	NA	86	426	539
	range	NA	ND-6.0	NA	ND-19	NA	NA	6-23	NA	12-176	98-679	122-879
1 Yusho baby from Taiwan (Rappe et al., 1983b)		NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
9 samples from exposed North Vietnamese 56% lipid (average) (Commoner et al., 1986)		ND	NA	0.42	NA	NA	NA	7.6	19.2	NA	92.4	NA
15 samples from exposed South Vietnamese 62% lipid (average) (Commoner et al., 1986)		22.3	NA	14.4	NA	NA	NA	99.8	178	NA	1326	NA
46 samples from exposed Americans prepared as composites from over 900 specimens 80% lipid (Commoner, et al., 1986)		6.3	NA	40	NA	NA	NA	90	110	NA	700	NA
3 samples from Vietnam veterans exposed to Agent Orange (average) (U.S. EPA 1985b)		20-173	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

Description	2,3,7,8-TetraCDD	Tetra CDD	1,2,3,7,8-PentaCDD	Penta CDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	Hexa CDD	1,2,3,4,6,7,8-HeptaCDD	Hepta CDD	Octa CDD	Total CDD
Adipose Tissue											
1 sample from Binghamton State Office building worker in New York after being exposed to CDD & CDF contaminated soot (Schecter et al., 1985a)	11.6	NA	15.0	NA	72.6	7.3	NA	209	NA	690	NA
4 people from New York that were unexposed (Schecter et al., 1985a)	3.7	NA	7.5	NA	60.4	6.8	NA	93.1	NA	586	NA
	6.0	NA	8.2	NA	60.3	7.4	NA	119	NA	695	NA
	7.2	NA	10.3	NA	54.5	7.5	NA	39.4	NA	593	NA
	8.3	NA	13.8	NA	46.2	7.4	NA	95.8	NA	534	NA
13 samples from a phenoxy acid sprayer who has been spraying for >10 years (average and standard deviation) (Nygren et al., 1985)	2±2.7	NA	6±2.5	NA	19±12	5±2.7	NA	104±93.5*	NA	98±207	NA
1 sample from exposed BASF worker who has had chloracne since 1953 (Nygren et al., 1985)	101	NA	18	NA	48	12.0	NA	20*	NA	80	NA
1 sample from chemist who has synthesized CDD and CDF isomers (Nygren et al., 1985)	NA	NA	5	NA	12	5	NA	100*	NA	374	NA
18 unexposed people (average) (Nygren et al., 1985)	3±2.0	NA	9±5.9	NA	12±3.9	4±1.0	NA	85±47*	NA	421±178	NA

APPENDIX D
TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION	2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
Adipose Tissue														
1 sample from Binghampton office building worker in New York after being exposed to CDD and CDF contaminated soot (Schecter et al., 1985a)	ND(2)	NA	NA	74.7	NA	149	112	NA	NA	39.3	25.9	NA	1.6	NA
4 people from New York that were unexposed (Schecter et al., 1985a)	ND(2)	NA	NA	16.5	NA	22.9	15.4	NA	NA	23.8	20.6	NA	1.5	NA
	ND(2)	NA	NA	17.0	NA	13.0	8.8	NA	NA	12.5	19.6	NA	1.2	NA
	4.1	NA	NA	10.9	NA	9.3	5.8	NA	NA	13.7	ND	NA	ND(20)	NA
	ND(2)	NA	NA	12.5	NA	11.4	5.6	NA	NA	16.3	ND	NA	ND(20)	NA
13 samples from phenoxy acid sprayers who have been spraying for >10 years (average and standard deviation) (Nygren et al., 1985)	4±2.1	NA	NA	50±23.6	NA	7±3.8	5±23.6	2±1.8	NA	14±12.4	NA	NA	<5	NA
1 sample from exposed BASF worker who has had chloracne since 1953 (Nygren et al., 1985)	<3	NA	NA	32	NA	11	5	2	NA	37	NA	NA	<50	NA
1 sample from chemist who has synthesized CDD and CDF isomers (Nygren et al., 1985)	7	NA	NA	26	NA	12	7	38	NA	17	NA	NA	240	NA
18 unexposed people (average & S.D.) (Nygren et al., 1985)	4±2.6	NA	NA	32±14.4	NA	5±1.5	4±1.4	2±1.0	NA	10±4.6	NA	NA	<5	NA

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

Description	2,3,7,8-TetraCDD	Tetra CDD	1,2,3,7,8-PentaCDD	Penta CDD	1,2,3,6,7,8-HexaCDD	1,2,3,7,8,9-HexaCDD	Hexa CDD	1,2,3,4,6,7,8-HeptaCDD	Hepta CDD	Octa CDD	Total CDD
1 sample from deceased Yusho patient (Masuda and and Yoshimura, 1984)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Liver Tissue											
1 deceased Yusho Patient (Masuda and Yoshimura, 1984; Rappe et al., 1983b)	2	NA	<2	NA	4	NA	NA	72	NA	350	NA
1 Yusho baby from Taiwan (Rappe et al., 1983b)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Blood											
Workers exposed to chlorophenol at a sawmill (Rappe et al., 1983b)	NA	NA	NA	NA	NA	NA	NA	<2	NA	5	NA
1 sample taken in Seveso, Italy after accident (Facchetti et al., 1980)	6	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Milk											
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	3	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Milk Lipid											
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	100	NA	NA	NA	NA	NA	NA	NA	NA	170	NA

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION	2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
1 sample from deceased Yusho patient (Masuda and Yoshimura, 1984)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	A 6000	13000
Liver Tissue														
1 deceased Yusho patient (Masuda and Yoshimura, 1984; Rappe et al., 1983b)	NA	<1	NA	10	NA	NA	NA	NA	55	100	NA	NA	<3	3000-25000
1 Yusho baby from Taiwan (Rappe et al., 1983b)	60	NA	194	91	NA	193	NA	NA	NA	NA	NA	NA	NA	NA
Blood														
Workers exposed to chlorophenol at a sawmill (Rappe et al., 1983b)	NA	NA	NA	NA	NA	NA	NA	NA	NA	40	NA	<1	<3	NA
1 sample taken in Seveso, Italy after accident (Facchetti et al., 1980)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION		2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
7 Swedish workers exposed to phenoxy acids (Hardell et al., 1985)	mean	NA	4.2	NA	NA	56	NA	NA	NA	19	NA	NA	18	NA	98
	range	NA	2.1-7.2	NA	NA	22-87	NA	NA	NA	8-35	NA	NA	5-49	NA	39-17
18 unexposed workers (Hardell et al., 1985)	mean	NA	4.2	NA	NA	32	NA	NA	NA	11	NA	NA	10	NA	57
	range	NA	0.3-11.4	NA	NA	9-65	NA	NA	NA	13-17	NA	NA	1-18	NA	13-10
1 Yusho baby from Taiwan (Rappe et al., 1983b)		17	NA	44	68	NA	88	NA	NA	NA	NA	NA	NA	NA	NA
9 samples from exposed North Vietnamese (average) 56% lipid (Commoner et al., 1986)		ND	NA	NA	9.8	NA	NA	NA	NA	10.3	2.3	NA	NA	ND	142
15 samples from exposed South Vietnamese (average) 62% lipid (Commoner et al., 1986)		ND	NA	NA	21.0	NA	NA	NA	NA	58.3	28.9	NA	NA	ND	1749
46 samples from exposed Americans prepared from over 900 specimens 80% lipid (Commoner et al., 1986)		11	NA	NA	34	NA	NA	NA	NA	22	22	NA	NA	75	1110
3 samples from Vietnam Veterans exposed to Agent Orange (average) (U.S. EPA, 1985b)		NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

Analyses of the rice oil indicated that over 40 CDF congeners were present ranging from tri to hexaCDF. Table D.4 shows the concentrations of CDFs detected by Rappe et al. (1983b) and Masuda et al. (1984) in the adipose tissue and liver of a "Yusho" baby from Taiwan and in the adipose tissue of a deceased "Yusho" patient. In the liver sample the dominant congener was 1,2,3,7,8-pentaCDF, while in adipose tissue the highest value was found for 2,3,4,7,8-pentaCDF.

A recent U.S. EPA survey of 46 pooled tissue samples taken from 900 individuals believed to be representative of the general U.S. population showed CDDs and CDFs are generally present (Commoner et al., 1986). Comparable data for North and South Vietnamese samples are also reported. The levels in North Vietnam are about an order of magnitude below those found in either South Vietnam or the United States. It is believed by Commoner et al. that the concentrations of CDD and CDF in adipose tissue of South Vietnamese (higher than in the United States samples by a factor of 3.5) is indicative of the exposure to Agent Orange. The CDD and CDF concentrations in Americans are believed by the authors to probably originate from (1) CDD and CDF contaminated chemicals that enter the food chain from waste effluents or agricultural sprays, and (2) combustion of chlorine-containing fuels emitting particles that eventually enter the food chain or get inhaled directly.

ENVIRONMENTAL CONTAMINATION--CALIFORNIA

Monitoring for CDDs and CDFs has not been as extensive in California as it has been in other areas. Much of the data produced to date and presented in the previous section has been related to industrial, occupational, and waste disposal practices that have caused environmental contamination and/or human exposure. Most of the industrial production of herbicides, such as 2,4,5-T, and of chlorophenol products containing CDDs and CDFs as contaminants, has been in other states which has helped to minimize the occurrence of contamination in California. Waste products contaminated with CDDs and CDFs from the manufacturing of these products have also been less of a problem, although there are other chemical production processes (see Appendix B) which may produce them as byproducts, some of which are in use by the chemical industry in California.

CDDs and CDFs chlorinated in the 2,3,7, and 8 positions have recently been evaluated and recommended for classification as toxic air contaminants by the Air Resources Board in a joint effort with the Department of Health Services (CARB and CDHS, 1986). While no monitoring has been conducted to date, estimates of emission factors indicate that combustion sources such as solid waste incinerators may provide a significant contribution to the CDD and CDF input into the environment (CARB and CDHS,

APPENDIX D

TABLE D.4

CDD AND CDF CONCENTRATIONS (ppt) IN HUMAN TISSUE

DESCRIPTION	2,3,7,8 Tetra-CDF	Tetra-CDF	1,2,3,7,8 Penta-CDF	2,3,4,7,8 Penta-CDF	Penta-CDF	1,2,3,4,7,8 Hexa-CDF	1,2,3,6,7,8 Hexa-CDF	2,3,4,6,7,8 Hexa-CDF	Hexa-CDF	1,2,3,4,6,7,8 Hepta-CDF	1,2,3,4,7,8,9 Hepta-CDF	Hepta-CDF	Octa-CDF	Total CDF
Milk														
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Milk Lipid														
5 samples from South Vietnam in 1973 (average) (Commoner et al., 1986)	NA	NA	NA	10	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

NA = Not Available
ND = Not Detected
(Numbers in parentheses are limits of detection)
< = less than
* = reported as 1,2,3,4,7,8,9-heptaCDD

TABLE D.5

CDD AND CDF CONCENTRATIONS IN CALIFORNIA FRESHWATER FISH AND SEDIMENT

Sample and Source	CDD (ppt)					CDF (ppt)				
	Tetra	Penta	Hexa	Hepta	Octa	Tetra	Penta	Hexa	Hepta	Octa
Carp Santa Ana River (U.S. EPA, 1986)	4.6[a]	-[b]	-	-	-	-	-	-	-	-
Sediment Mill Creek (U.S. EPA, 1986)	0.6[a]	-	-	-	-	-	-	-	-	-
Striped Bass Sacramento River (USFWS, 1983)	ND[c]	ND	ND	ND	ND	10	9	TR[d]	2	4.5
Striped Bass Eggs Sacramento River (USFWS, 1983)	6[a]	ND	ND	ND	ND	30	4	ND	ND	ND

a/ 2,3,7,8 Isomer
b/ Not Analyzed (-)
c/ Not Detected (ND)
d/ Trace (TR)

1986; Wong, 1984). Anywhere from one to ten percent of the chlorophenols used in wood treatment operations may be burned with wood wastes (Bridle et al., 1984).

Hazardous waste site investigation and cleanup activities, which include wood treatment facilities, have provided evidence of CDD and CDF contamination in the State. The U.S. EPA National Dioxin study and a U.S. Fish and Wildlife study have demonstrated the presence of these toxicants in a limited number of fish and river sediment samples. The U.S. EPA indicates more monitoring in California for CDDs and CDFs related to the use of chlorophenols in the wood industry is likely in the future (U.S. EPA, 1986f).

Fish and Freshwater Sediment Studies

The U.S. Fish and Wildlife Service (1983) conducted a study in 1980 to 1981 to assess the effect of various water contaminants on striped bass populations. The study involved striped bass adults, young and eggs. While most samples were taken from rivers in the eastern U.S., the Sacramento River was included on the west coast, with analysis for all Cl_4 to Cl_8 CDDs and CDFs. The results, shown in Table D.5, indicate more CDFs than CDDs present in both whole fish and in fish eggs, with tetra- and pentaCDFs detected at higher levels than the higher chlorinated isomer groups. 2,3,7,8-TetraCDD was found only in the eggs, with 2,3,7,8-tetraCDF present in both whole fish and in the eggs.

As part of the National Dioxin Study by the U.S. EPA (1986e), samples were taken of fish and sediment from the Santa Ana River in southern California. This river was selected because it receives industrial and agricultural inputs from many sources. This study looked only for 2,3,7,8-tetraCDD, which was found in carp at a level of 4.6 ppt, and in a sediment sample from Mill Creek at a level of 0.6 ppt near its confluence with the Santa Ana River.

SUMMARY AND DISCUSSION

CDDs and CDFs are formed as contaminants from precursor compounds during the production of certain chemical products, and are released to the environment in the course of product use or disposal. Chemical products include, but are not limited to phenoxy herbicides, PCBs, diphenyl ether herbicides, and chlorophenol wood preservatives. CDD and CDF contaminated wastes generated in the manufacture and use of these products have also caused serious contamination through the use of unsound disposal practices.

Fish in several areas of the U.S. and other parts of the world have levels of CDDs and CDFs which have resulted in health advisories regarding consumption. Waste disposal practices and industrial process effluents are believed to be the sources of contamination.

APPENDIX E

OVERVIEW OF ANALYTICAL METHODOLOGY

In Vietnam, the use of Agent Orange, a mixture of the phenoxy herbicides 2,4-D and 2,4,5-T containing CDDs and CDFs at low ppm levels as contaminants, has resulted in elevated levels of CDDs and CDFs in both humans and the environment. Similar studies of phenoxy herbicide use, especially 2,4,5-T in this country and in others, have determined product-related environmental and human residues.

Human exposure has resulted from several accidents, industrial and otherwise, which have caused some of the more significant contamination episodes. The evacuation of the town of Times Beach, Missouri due to the use of CDD and CDF contaminated oil for dust control on roads, the chemical explosion in Seveso, Italy in 1976 which caused large-scale contamination, and the ingestion of rice bran oil contaminated with CDFs (Yusho poisoning) in Japan and Taiwan are some of the more serious events affecting large numbers of people.

Several studies have determined the presence of CDDs and CDFs in tissues from the general population, indicating a probable steady background exposure. These compounds have also been found in places far from discrete sources. Combustion of CDD and CDF containing wastes and wastes which contain precursor materials capable of forming them, is believed to be a significant source and may be responsible for their ubiquitous presence in the environment. The California Air Resources Board has recently recommended that CDDs and CDFs be classified as toxic air contaminants, based in part on an evaluation of adverse health effects conducted by the California Department of Health Services.

In California, CDDs and CDFs have been determined as contaminants during investigations of hazardous waste sites, many of which are locations of present and former wood treatment operations. Levels have also been found in limited fish and river sediment samples in the State.

APPENDIX E

OVERVIEW OF ANALYTICAL METHODOLOGY

Analysis for CDDs and CDFs in environmental or biological samples is a challenging and difficult task for a number of reasons. There are 75 CDD and 135 CDF congeners with very similar physical and chemical characteristics. Many other commonly occurring compounds can interfere with the analysis. The analytical methods used must be able to separate, identify, quantify, and confirm the identity of the various congeners (Rappe et al., 1983a), especially those chlorinated in the 2,3,7, and 8 positions of toxicological concern. The major components of such a method are (Tiernan, 1983):

- extraction of CDDs and CDFs from the sample matrix;
- cleanup of the extract to isolate the CDDs and CDFs from potentially interfering compounds; and
- detection, identification, and quantification.

Extraction solvents used include hexane, hexane-acetone, benzene, toluene, chloroform, and methylene chloride (Esposito et al., 1980). The selection of extraction solvent is sample matrix dependent, and in some cases acid/base digestion may also be required when working with biological tissues or soil samples where CDDs and CDFs may be tightly bound to the sample matrix. Various means of extraction may be needed to ensure efficient extraction of CDDs and CDFs from different sample matrices, and where shaking or ultrasonification may serve for one media, soxhlet extraction or other rigorous methods may be necessary for others (U.S. EPA, 1985a).

Cleanup of the sample extract is required prior to instrumental analysis. The primary goal is to remove any co-extracted lipids or other chlorinated hydrocarbons which may interfere with analysis by coeluting on the GC column and/or by producing ions of the same mass as the CDDs and CDFs in the mass spectrometer. Extraction solvents which include hexane, chloroform, hexane-acetone, petroleum ether and chloroform-methanol may be used, and washing the extract with acid or base may also be required depending on the nature of the lipids present, with acid preferred to avoid decomposition of the CDDs and CDFs. Other impurities such as pesticides are removed by liquid chromatography using adsorbents including alumina, Florisil, charcoal and silica (U.S. EPA, 1985a). Additional cleanup may involve the use of high performance liquid chromatography (HPLC) and/or liquid column chromatography to remove other interfering compounds, or fractionation of the extract prior to GC-MS analysis for better isomer resolution (Esposito et al., 1980).

Recovery of the internal standards added to the sample just prior to extraction and analysis may not be a reliable indicator of the extraction and analysis for the CDDs and CDFs contained as

for 2,3,7,8-tetraCDD to be similar for both high and low resolution instruments, with high resolution instruments better able to resolve interfering compounds.

After separation of the GC column, the compound is fragmented in the mass spectrometer, producing ions with both the electron impact (EI) and negative chemical ionization (NCI) modes giving good results with CDDs and CDFs. These ions are separated by their mass:charge ratios (m/z), and in most cases are detected by an electron multiplier. The ions produce a fragmentation pattern that is characteristic of the compound of interest, although positional isomers within an isomer group may have simlar patterns. The occurrence of the fragmentation pattern at particular energies and times is the mass spectrum for a compound (Rose and Johnstone, 1982). The signal generated is a measure of the number of ions producing it, which may be used quantitatively when compared with that of the internal standard (Esposito et al., 1980). While there may be many ions making up the mass spectrum of a compound, high sensitivity is achieved by selectively monitoring only a small number of ions that are characteristic of the compound of interest, usually 1-4 ions. This is known as selected ion monitoring (SIM) or multiple ion detection (MID) (Buser et al., 1985; Tiernan, 1983; Esposito et al., 1980). However, some specificity is lost because the entire mass spectrum is not obtained.

Identification and quantification of individual congeners is based on the retention time of the compound on the GC column, and the mass ratio of the compound in the sample compared to that of a standard reference compound. Internal standards are required during CDD and CDF analysis, and both ^{37}Cl and ^{13}C isotope labeled standards and unlabeled standards are added to the sample in known amounts prior to analysis. Such standards are useful for determining retention times on the GC column, and also for quantification of the CDDs and CDFs in the sample based on the instrument response factor for a particular congener.

A study by Rappe et al. (1983a), using tetra- through octaCDF congeners demonstrated significant differences in the response factors between isomers when equal amounts were analyzed by GC-MS. Response factors in the election impact mode varied by about 1.6 for the tetraCDF isomers, 2.5 for pentaCDF isomers and by 1.5 for hexaCDF isomers when peak areas were compared. For reliable quantitative analysis of individual isomers, it is apparent that response factors obtained from actual isomer standards should be used, and not estimates obtained from other isomers (Rappe et al., 1983a; Rappe, 1984; U.S. EPA, 1985a). Unfortunately, reference standards for all congeners of interest are not currently available, which lends uncertainty to results based on other congeners. This situation should improve as more standards become available.

contaminants in soil and sediment sample matrices (U.S. EPA, 1986c). For such matrices there seems to exist an inverse relationship between the length of time the CDDs and CDFs are in contact with the soil or sediment and their extractability.

The result of extraction and cleanup is concentration of CDDs and CDFs and removal of as many interferences as possible. Potentially interfering compound classes include (U.S. EPA, 1985a; Smith et al., 1983):

- PCBs
- naphthalenes
- diphenyl ethers
- methoxy-PCB
- hydroxy-PCB
- hydroxy diphenyl ethers
- benzylphenyl ethers
- biphenylenes
- DDT (4 isomers)
- DDE (4 isomers)

Although other methods including gas chromatography - electron capture (GC-ECD) and UV spectroscopy have been used in the past for CDD and CDF analysis, gas chromatography - mass spectrometry (GC-MS) is the method of choice in use today (Tiernan, 1983). The most common system configuration combines high resolution gas chromatography with low resolution mass spectrometry (HRGC-LRMS), and was the method used by both State Board contract labs for congener-specific analysis. Details of the analytical methodologies used by the contract labs are included in Appendices F and G.

High resolution gas chromatography is used as a means of identifying individual congeners by resolving (separating) them from each other through the use of a fused silica or glass capillary column coated with an appropriate stationary phase (Rappe, 1983). There are various GC columns suitable for CDD and CDF analysis, and the choice of which to use may depend on which congeners are of interest. In some cases isomers that coelute on one column may be resolved on another, and different columns may be used in combination if necessary (U.S. EPA, 1985A). Capillary columns which have proved useful are OV-17, OV-101, Silar 10C, SE 30, SP-2330, SP-2331 and DB-5 among others (Crummett, 1983). Other advantages of capillary columns in GC-MS are: the narrow band width of the eluting compounds allows for increased sensitivity in the mass spectrometer; the low column bleed rates minimize background contamination; and mixed isomers and interfering compounds are resolved more efficiently (U.S. EPA, 1985a).

While there are high resolution GC-MS methods available (resolution of at least 1:10,000), low resolution instruments are more commonly used, and provide both the specificity and sensitivity required for CDD and CDF analysis, especially when coupled with high resolution capillary GC (Tiernan, 1983). Although high resolution instruments are capable of lower detection limits by factors of 10-100 when compared to low resolution instruments, Crummett (1983) found detection limits

APPENDIX F

ANALYTICAL METHOD SUMMARY

California Analytical Laboratories
Sacramento, California

California Analytical Laboratories, Inc.
2544 Industrial Boulevard • West Sacramento, CA 95691 • (916) 372-1393

April 8, 1986

Frank Palmer
State Water Resources Control Board
P.O. Box 100
Sacramento, CA 95801

Dear Mr. Palmer;

Please find enclosed a brief description of our method for identifying and quantitating C14 to C18 Dioxin and Furan samples.

Identification is based on two criteria, retention time and m/e ratio. If the mass chromatograms have the proper ratio then that peak is identified as a possible native constituent. If this peak lies within predetermined retention time windows, the peak is positively identified. The area is then added to all others that meet the same criteria, and this is the total value used in calculating the amount found.

If 2,3,7,8 substituted isomers of C14 to C17 are desired, the criteria above are followed, but on two columns (DB-5 and SP-2331) instead of just the DB-5 alone. For the 2,3,7,8-substituted compounds exact retention times ($\pm$0.001 RT units) are known from the standard runs and calculated. Thus if a peak has the proper RRT and ratio on the DB-5 column, it is a tentative 2,3,7,8-substituted isomer. The sample is then confirmed on SP-2331 which chromatographs the compounds differently. Again, using standard RRT and ratios, if the peak in the samples agrees with the standards it is confirmed as a positive 2,3,7,8 substituted isomer.

If this dual agreement is not met, the compound can not be identified solely as a 2,3,7,8 substituted isomer, but may in fact be another isomer of the same congener.

This dual column confirmation is required with RRT and m/e ratios agreeing with daily standard runs to identify a peak as a 2,3,7,8 substituted dioxin or furan isomer.

If there are any questions, please do not hesitate to call.

Sincerely,

Michael J. Miille, PhD
Director of GC/MS Services

Michael W. Orbanosky
GC/MS Lab Manager

dlc

STANDARDS AND REAGENTS.

The [13C]-2378-TCDD (ED-900), [37Cl]-2378-TCDD (ED-907), [13C]-12378-PeCDD (ED-955), [13C]-123678-HxCDD (ED-966), [13C]-1234678-HpCDD (ED-972), [13C]-OCDD (ED-981), [13C]-2378-TCDF (EF-904), 2378-TCDD (ED-901), 12378-PeCDD (ED-950), 123678-HxCDD (ED-961), 1234678-HpCDD (ED-971), OCDD (ED-980), 2378-TCDF (EF-903), 12378-PeCDF (EF-953), 123678-HxCDF (EF-962), 1234678-HpCDF (EF-973), OCDF (EF-982) were all obtained from Cambridge Isotope Laboratories, Inc. The solvents used were methylene chloride (Baker #9264), hexane (Baker# 9262), methanol (Fisher #A-936), toluene (Baker #9336), and tetradecane (Aldrich #17,245-6). Other reagents used were sodium sulfate (Malinkrodt #8024), silica gel (Kieselgel 60, EM Reagents #7734), basic alumina (Bio-Rad AG-10, stored at 130 C after being kilned at 600 C for 12 hours), and acid alumina (Bio-Rad AG-4) stored at 130 C. The 44% H_2SO_4/silica gel was prepared by adding 44 grams of sulfuric acid (Baker #9681) to 56 grams of silaca gel and mixing well. The 33% 1N NaOH/silca gel was prepared by adding 33 grams of 1N NaOH (Baker #3727) to 67 grams of silca gel and mixing well; these two packings were stored at ambient in closed containers until used. The carbopack/silca gel packing was prepared by mixing 3.6 grams of carbopack (Supelco #1-0257) and 16.4 grams of silica gel and storing at 130 C.

EXPERIMENTAL

Soil/solid sample extraction.
The sample was weighed to two significant figures into a 250ml erlenmeyer flask. See Chart I for sample size. Internal standards were then added to the sample (see Chart II) and if appropriate, the native spike compounds added (see Chart II). After adding 20 grams of sodium sulfate, 20ml of methanol, and 150ml of hexane, the flask was placed on a shaker for 3 hours. The extract was filtered and set aside for clean up.

Liquid/aqueous sample extraction.
A measured volume of sample was transfered to a separatory funnel. See Chart I for sample size. Internal standards were then added to the sample (see Chart II) and if appropriate, the native spike compounds added (see Chart II). Distilled water was added to dilute the sample matrix and aid in the extraction. The sample/water mixture was extracted three times using fresh 50ml portions of methylene chloride. These extracts were combined and set aside for clean up.

Extract clean up.
If the extract was suspected to have high levels of organics, the extract was transfered to a separatory funnel and washed with a 50ml portion of 10N NaOH. The extract was subsequently washed with 50ml of distilled water, two 25ml portions of sulfuric acid and 50ml of distilled water. The washed extract was then passed through sodium sulfate into a 250ml round bottom flask containing approximately 500ul tetradecane and roto-evaporated to the

California Analytical Laboratories, Inc.
2544 Industrial Boulevard • West Sacramento, CA 95691 • (916) 372-1393

July 24, 1986

Frank Palmer
State Water Board
P.O. Box 100
Sacramento, CA 95801

Dear Frank:

Enclosed is a copy of the method description for our dioxin and furan analyses of the PCP samples submitted earlier this year. We have tried to provide the detail in a form similar to a publication as Jerry Bowes requested.

If you need any additional information, please give me a call.

Sincerely,

Michael J. Miille, PhD
Vice President

jb

CHART II

Internal Standards	
^{13}C-2378-TCDD	25ng
^{13}C-2378-TCDF	25ng
^{13}C-12378-PeCDD	50ng
^{13}C-123678-HxCDD	50ng
^{13}C-1234678-HpCDD	50ng
^{13}C-OCDD	250ng
(^{37}Cl-2378-TCDD	10ng)

Native Spike Compounds
2378-TCDD
12378-PeCDD
123678-HxCDD
1234678-HpCDD
OCDD
2378-TCDF
12378-PeCDF
123678-HxCDF
1234678-HpCDF
OCDF

tetradecane and set aside for clean up.

If the expected level of organics of the original extract was low, the acid/base clean up was by-passed. Approximately 500ul of tetradecane was added to the extract and the extract roto-evaporated to the tetradecane (after removal of residual water by passing the extract through sodium sulfate) and set aside for clean up.

The concentrated extract was cleaned up by the use of a four column system. Column 1 (1.5cm x 40cm) was packed by adding 1 gram of silica gel followed by 2 grams of 33% 1N NaOH/silica gel, 1 gram silica gel, 4 grams of 44% H_2SO_4/silica gel, 2 grams of silica gel and 1 gram of sodium sulfate to the column. Column 2 was packed with 6 grams of acid alumina. Columns 1 and 2 were rinsed with hexane and Column 1 placed above Column 2. The concentrated extract was transfered to Column 1 with small portions of hexane. Column 1 was eluted with 90ml of hexane directly onto Column 2 and then discarded. Column 2 was eluted with 20ml hexane followed by 20ml of 20% methylene chloride/hexane. Approximately 500ul of tetradecane was added to the methylene chloride/hexane eluate and concentrated under nitrogen to the tetradecane. The concentrated extract was transfered to Column 3 (1.1cm x 30cm column packed with 5 grams of basic alumin on top of 1 gram of sodium sulfate and rinsed with hexane) with small portions of hexane. Column 3 was eluted with 20ml of hexane, which was discarded, followed by 8ml of 3% methylene chloride/hexane, which was archived. Next Column 3 was eluted with 35ml of 50% methylene chloride/hexane . After adding approximately 500ul of tetradecane, the eluate is concentrated under nitrogen to the tetradecane. Column 4 (5ml graduated dispo-pipet) contains 0.3 grams of carbopack/silica gel packed between two glasswool plugs. The column was rinsed with 5ml of hexane, then turned over and rinsed sequentially with 5ml of hexane, 2ml of toluene, 1ml of 75:20 methylene chloride/methanol, 1ml of 1:1 methylene chloride/cyclohexane and 2ml of hexane. The concentrated extract was transfered with small portions of hexane to the column and eluted sequentially with two 1ml portions of hexane, 1ml of 1:1 methylene chloride/cycloheaxne, and 1ml of 75:20 methylene chloride/methanol. These eluates were discarded and the column turned over and eluted with 4ml of toluene. Tetradecane (50ul) was added to the toluene eluate and the toluene removed by nitrogen. This concentrated, cleaned up extract was stored in the dark until GC/MS injection.

GC/MS Analysis

The GC columns used were a DB-5 60M X 0.32mm ID fused silica column (J&W Scientific, Ranch Cordova) and a SP-2331 60M X 0.32mm ID fused silica column (Supelco, Belefonte). See Table I for GC temperature programs used for each column.

Mass spectral analysis was performed using a Finnigan MAT 5100 gas chromatograph mass spectrometer with a Nova 4X data system in SIM mode. The instrument was calibrated daily using FC-43 (perfluorotributylamine). After this initial calibration the SIM tables were updated against the new calibration table. Because of the number of masses monitored, two sets of SIM descriptions were used in sequence. See Table II for description of each SIM set. Calibration standards equivalent to 1 ppb to 25 ppb (0.2 ng/uL to 5 ng/uL) were prepared from stock solutions. The following working standards were used to establish average response factors for each of the dioxin and furan homologs.

0.2 ng/uL	Cl_4 - Cl_7	D/F
0.5 ng/uL	Cl_8	D/F
1.0 ng/uL	Cl_4 - Cl_7	D/F
2.0 ng/uL	Cl_8	D/F
5.0 ng/uL	Cl_4 - Cl_7	D/F
5.0 ng/uL	Cl_8	D/F

The relative response factors (RRF) for both the native D/F and surrogate were calculated using equations 1 and 2.

Equation 1 (RRF for native D/F)

$$RRF = A_S\ C_{IS}/A_{IS}\ C_S$$

A_S = Area Native Std.
A_{IS} = Area Int. Std.
C_S = Conc. Native Std.
CIS = Conc. Int. Std.

Equation 2 (RRF for $^{37}Cl_4$-TCDD surrogate)

$$RRF = A_{SS}\ C_{IS}/A_{IS}\ C_{SS}$$

the 328 response is corrected by subtracting 0.0009 of the 322 response.

A_{SS} = Area Surrogate Std.

C_{SS} = Conc. Surrogate Std.

The masses monitored for the calculations are shown below.

Homolog	Native Standard m/e	Int. Std. m/e
Cl_4 - F	306	318
Cl_4 - D	322	334
$^{37}Cl_4$ -D	328	334
Cl_5 - F	342	370
Cl_5 - D	358	370
Cl_6 - F	376	404
Cl_6 - D	392	404
Cl_7 - F	410	438
Cl_7 - D	426	438
Cl_8 - F	444	472
Cl_8 - D	460	472

The RRF over the working range should be constant (40% RSD). If this criteria was met then the average RRF was calculated for each homolog and used in calculating the results. The RRF for native compounds and surrogate were verified every 12 hours or less by calculating the RRF on one or more (usually the 1 ng/uL) standards run during the course of the 12 hour period. A working standard was run at the end of every 12 hour period. If the variation of the RRF's was greater than $\pm$ 10% a new curve was run.

Initially, sample extracts are analyzed using the DB-5 column. This column can be run at a higher temperature, resulting in shorter sample runs and better chromatography for the hepta and octa chlorinated dioxins and furans The total Cl_4, Cl_5, Cl_6, Cl_7 and Cl_8 concentrations can be calculated as follows. Quantitative requirements must be met before a peak can be identified as a native congener. The monitored mass ratio must be within $\pm$ 10% of the standard mass ratio. The masses monitored must maximize together and have a signal to noise ratio greater than 2.5 to 1. All peaks must elute within the previously established retention time window. If these criteria were met then the areas for all the peaks for a homolog were summed and this value (A_S) used in equation 3 to calculate the total concentration of that homolog.

Equation 3 $C_S = A_S\ C_{IS}/A_{IS}$ X RRF_S X wt sample.

If quantitation of the 2,3,7,8-substituted isomers was required the sample extracts and appropriate standards were injected on the SP-2331 FSCC.

TABLE I

Recommended GC Capillary Conditions

Column	60 meter (SP 2331)	60 meter DB-5
2,3,7,8-TCDD R.T.	12 min	10.0 min
Helium Linear Velocity		30 cm/sec
Initial Temperature	190 C	190 C
Initial Time	1 min	1 min
Splitless Time	0.6 min	0.5 min
Program Rate	10 C/min	8 C/min
Final Temperature	250 C	300 C
Final Hold Time	15 min	7 min
Split Flow	30 ml/min	30 ml/min
Septum Purge Flow	5 ml/min	5 ml/min
Capillary Head Pressure	28 psi	15 psi

The above GLC conditions are employed for both classification and the isomer specific analysis of tetra through octa chlorodioxins and dibenzofurans.

The 60 meter DB5 is appropriate for total Cl_4 - Cl_8 D/F classification. Isomer specific analysis of all 2,3,7,8-substituted chlorinated dibenzodioxins and dibenzofurans required a 60 meter DB-5 and a 60 meter SP 2331.

Column isomer specificity was checked using a mix that includes 2378, 1478, 1234, 1237, 1238, 1278 and 1267 TCDD. The column must separate 2,3,7,8-TCDD from the remaining TCDD isomers with at least a 25% valley. It should also separate 12348 and 12378 PnCDF, and 123478 and 123678 HxCDD with at least a 60% valley. Qualitative identification requires that the m/e ratios for each congener are within ± 10% of that for the standard. The signal to noise must be 2.5/1. The relative retention time must be within ± 0.005 units for the standard RRT. These criteria must be met to positively identify the 2378 isomer. If these criteria were met the isomer concentrations were calculated using equation 3. When an isomer gave a positive value on both the DB-5 and SP-2331 column, it was considered positive and the quantitative value from the SP-2331 column was reported.

APPENDIX G

ANALYTICAL METHOD SUMMARY

IIT Research Institute
Chicago, Illinois

TABLE II
Selected Data for the Polychlorinated Dibenzofurans
and
Polychlorinated Dibenzo-P-Dioxins of Interest

Compounds	Accurate Mass		Theoretical Isotope Ratio
	Low Mass	High Mass	
Di-CDF	235.980	237.977	1.5
Di-CDD	251.974	253.972	1.5
Tri-CDF	269.941	271.938	1.01
Tri-CDD	285.936	287.933	1.01
Tetrachlorodibenzofuran	303.9016	305.8987	0.77
Tetrachlorodibenzo-p-dioxin	319.8965	321.8936	0.77
$^{13}C_{12}$-2,3,7,8-TCDD	331.9368	333.9339	0.77
$^{13}C_{12}$-2,3,7,8-TCDF	317.9390	319.937	0.77
Pentachlorodibenzofuran	339.8597	341.8567	1.54
Pentachlorodibenzo-p-dioxin	355.8546	357.8517	1.54
$^{13}C_{12}$-12378-PnCDD	367.8947	369.8918	1.54
$^{13}C_{12}$-12378 or 23478-PnCDF	351.9000	353.8970	1.54
Hexachlorodibenzofuran	373.8207	375.8178	1.23
Hexachlorodibenzo-p-dioxin	389.8156	391.8127	1.23
$^{13}C_{12}$-HCDF	385.8610	387.8581	1.23
$^{13}C_{12}$-HCDD	401.8559	403.8530	1.23
Heptachlorodibenzofuran	407.7817	409.7788	1.03
Heptachlorodibenzo-p-dioxin	423.7766	425.7737	1.03
$^{13}C_{12}$-Hepta-CDD	435.8169	437.8140	1.03
$^{13}C_{12}$-Heptachlorodibenzofurans	419.8220	421.8191	1.03
Octachlorodibenzofuran	441.7428	443.7398	0.88
Octachlorodibenzo-p-dioxin	457.7377	459.7347	0.88
$^{13}Cl_{12}$-Octa-CDD	469.7780	471.7750	0.88
$^{13}C_{12}$-Octachlorodibenzofuran	453.7831	455.7801	0.88

IIT Research Institute
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312/567-4000 • Telex 282472 IITRICHGO

June 30, 1986

Mr. Francis Palmer
State Water Resources Control Board
Paul R. Bonderson Bldg.
901 P Street
Sacramento, CA 95801

Dear Mr. Palmer:

Enclosed you will find the additional information you requested. I have provided a detailed description of both the extraction and analytical procedures used to analyze the eight samples (23446 - 1 through 8) for PCDDs and PCDFs. In conjunction with the analytical description, I have included a table listing the analytical sequence (masses monitored, elution windows, etc.) used during the GC/MS analyses, as well as, selected ion current chromatograms (Figure) displaying the elution sequence of the 2,3,7,8-substituted dioxin and furan isomers on the SP2330 capillary column. Also enclosed are the selected ion current profiles for each sample and the updated data sheets, incorporating the revised OCDF concentrations and calculated detection limits. The detection limits were determined for a specific isomer within each chlorination level (tetra, penta, etc.) and then applied to each PCDD/PCDF congener class.

If you have any questions regarding the enclosed information or are in need of any additional information, please contact the undersigned at 312/567-4234.

Respectfully submitted,
IIT Research Institute

Michael Miller

Michael Miller
Research Chemist
Chemistry Research Section

Approved

D. J. Moschandreas, Ph.D.
Research Director
Chemistry and Chemical Engineering
Research Department

1.2.3 To determine the sample weights triplicate weight measurements were made on the appropriate sample volumes. The average of these three determinations was taken as the sample weight.

1.2.4 Prior to adding the internal standard spiking solution (Section 1.1.4) to the sample, 100 µl of this solution was mixed with 1 ml of acetone. This was done in order to enhance mixing of the spiking solution with the predominantly water matrix. (Due to an oversight, this mixing step was omitted for samples 23446-4 and 5. For these two samples the spiking solution was added directly to the sample-water mixture.)

1.2.5 The sample was extracted three times with 60 ml volumes of methylene chloride.

1.2.6 The combined methylene chloride extracts were concentrated by roto-evaporation to approximately 1 ml. To the concentrator flask, 50 ml of hexane were added and the volume again reduced to 1-2 ml.

1.2.7 The sample extract was transferred to a 125-ml separatory funnel using four 10-ml aliquots of hexane.

1.2.8 The hexane sample-extract was initially washed with 50 ml of 10N sodium hydroxide. The aqueous layer was discarded.

1.2.9 The organic layer was then washed with 50 ml of reagent water, which was also discarded.

1.2.10 The hexane layer was washed with two 50-ml aliquots of sulfuric acid. Both acid fractions were discarded.

1.2.11 The final extraction step consisted of two washings with 50-ml aliquots of reagent water. The aqueous fractions were again discarded.

1.2.12 The organic layer was then dried by swirling over 1-2 grams of anhydrous sodium sulfate.

1.2.13 The sample extract was again concentrated using roto-evaporation to approximately 1 ml prior to IFB-option A column cleanup.

1. SAMPLE EXTRACTION

1.1 SOLID SAMPLES

1.1.1 Each sample was opened, examined and its' physical appearance noted.

1.1.2 Samples were weighed on an analytical balance, which had been checked for accuracy by IITRI's QA/QC officer, and transferred into the extraction jar.

1.1.3 Purified anhydrous sodium sulfate (20 grams) was added to each sample and mixed thoroughly using a stainless steel spatula.

1.1.4 The internal standard spiking solution (100 µl) was then added directly to each sample. This solution contained: 250 ng/ml each of ^{13}C-2,3,7,8-TCDD and ^{13}C-2,3,7,8-TCDF; 500 ng/ml each of ^{13}C-1,2,3,7,8-PnCDF and ^{13}C-1,2,3,4,7,8-HxCDF; and 2200 ng/ml of ^{13}C-OCDD.

1.1.5 The spiked samples were then left to stand at ambient temperature. After two hours they were mixed and left for at least six more hours.

1.1.6 Methanol (20 ml) was added and the samples stirred. Then hexane (150 ml) was added and the samples were vigorously extracted for a minimum of three hours using a platform shaker.

1.1.7 The containers were removed from the shaker and the solids allowed to settle before proceeding. The extracts were carefully decanted through a glass funnel fitted with a solvent-rinsed filter paper. The extraction jar, its contents and the filter residue were carefully rinsed with hexane.

1.1.8 The extracts were concentrated using a Kuderna-Danish (K-D) evaporative concentrator in a water bath at 90°C. When the extract volume reached approximately 1 ml, the samples were ready for cleanup.

1.2 LIQUID SAMPLES

1.2.1 Each sample was opened, examined and its' physical appearance noted.

1.2.2 To facilitate the extraction of the three liquid samples each was mixed with one liter of reagent (Milli-Q) water. Sample volumes of 100 µl were used for samples 23446-4 and 5, and 10 ml for sample 23446-6. The samples were added to the reagent water which had been poured into two-liter separatory funnels.

2.2.2 The hexane sample extract was quantitatively transferred to the top of the sulfuric acid-impregnated silica gel, and the K-D concentrator tube was rinsed with 2 x 0.5 ml of hexane and placed onto the column.

2.2.3 The extract was then eluted directly onto the alumina column with 90 ml of hexane. The silica gel column was discarded.

2.2.4 Hexane (20 ml) was placed onto the alumina column and eluted until the liquid dropped below the sodium sulfate layer. The hexane eluant was discarded.

2.2.5 The alumina column was then eluted with 20 ml of 20% (v/v) methylene chloride/hexane solution. This solution was collected and concentrated to about 1 ml using a gentle stream of filtered nitrogen gas. A solvent exchange was then performed by adding 10 ml of hexane and reconcentrating to 1 ml. At this point the sample extract was ready for the final cleanup step.

2.3 BASIC ALUMINA COLUMN CHROMATOGRAPHY

2.3.1 A 1 cm x 30 cm chromatography column was dry-packed with 10.0 g of Fisher A-540 basic alumina and topped with 1 cm of purified sodium sulfate. The basic alumina had been activated at 130°C for a minimum of 16 hours.

2.3.2 The hexane sample extract from the Option A cleanup was transferred onto the bead of the A-540 column. The concentrator vessel was rinsed with 2 x 1 ml of 2% (v/v) methylene chloride - hexane solution and placed onto the column.

2.3.3 The A-540 column was then eluted with 80 ml of the 2% methylene chloride-hexane solution to remove such interferences as polychlorinated diphenyl ethers, benzenes, naphthalenes and biphenyls. This fraction was discarded.

2.3.4 The polychlorinated dioxins and furans were then eluted from the A-540 column with 120 ml of 20% (v/v) methylene chloride-hexane solution. This volume was concentrated to several milliliters by roto-evaporation and then quantitatively transferred to a concentrator tube where the volume was reduced to 1 ml using filtered nitrogen. The extract was then transferred to a 2-ml conical mini vial and the concentrator tube rinsed three times with the 20% methylene chloride-hexane solution. Between aliquot transfers the volume in the vial was gently reduced with nitrogen. When the volume reached 50-100 µl the walls of the mini vial were rinsed and the contents of the vial concentrated to near dryness. At that point the volume of the extract was adjusted to 50 µl with toluene in preparation for the GC/MS analysis.

2. SAMPLE CLEANUP

2.1 ACID/BASE EXTRACTION

In order to remove the anticipated high levels of pentachlorophenol (PCP), the extracts from the solid samples required an initial cleanup (described below) prior to the column chromatography. This acid/base cleanup step was incorporated in the extraction procedure for the liquid samples and was therefore unnecessary for these samples.

2.1.1 The organic extracts were washed with 30 ml of 20 percent aqueous potassium hydroxide by shaking for 10 minutes. The water layer was removed and discarded.

2.1.2 The extracts were then washed with 25 ml of reagent water by shaking for two minutes. The aqueous layer was removed and discarded.

2.1.3 To the organic extract, 50 ml of concentrated sulfuric acid was cautiously added. This mixture was shaken for 10 minutes and then allowed to stand until the aqueous and organic layers separated. The aqueous layer was removed and discarded. The acid washing was repeated until no color was visible in the acid layer.

2.1.4 The organic extracts were washed with reagent water (20 ml) for two minutes. The aqueous layer was removed and discarded. The organic layer was dried by adding 10 g of anhydrous sodium sulfate.

2.1.5 The extracts were then transferred to a K-D apparatus and concentrated to approximately 1 ml.

2.2 SILICA GEL/ALUMINA COLUMN CHROMATOGRAPHY (OPTION A)

2.2.1 A 1 cm x 30 cm chromatography column was packed with 1.0 g of silica gel,[1] 2.0 g sodium hydroxide-impregnated silica gel (33% w/w 1M NaOH), 1.0 g silica gel, 4.0 g of sulfuric acid-impregnated silica gel (40% w/w H_2SO_4) and 2.0 g silica gel. A second column (1 cm x 30 cm) was packed with 6.0 g alumina[1] and topped with a 1-cm layer of purified sodium sulfate. Hexane was added to both columns until the packings were free of channels and air bubbles.

---

[1]Silica gel (for column chromatography, type 60, EM Reagent, 100-200 mesh) and alumina (acid alumina, AG 4, BIO-RAD Laboratories) were Soxhlet-extracted with CH_2Cl_2 for 21 hours and activated at 130°C and 190°C, respectively, before use. Each batch was tested for proper recoveries prior to use.

standard as a reference. Since only a limited number of these standards are available in practice, the approach generally followed is to use an appropriate set of internal standards which includes representative isomers from each chlorinated class of PCDFs and PCDDs, and further assume that the data obtained for these is representative of all isomers in each group.

Relative response factors were determined from the analysis of standard solutions containing the internal standards and isomers representative of each chlorination class. Response factors were calculated from the following equation:

$$RF = Ax.Qis/Ais.Qx$$

where: Ax = sum of the integrated ion abundances of the masses for the unlabeled compound
Ais = sum of the integrated ion abundances of the masses for the appropriate labeled compound
Qis = amount of the appropriate labeled compound (ng)
Qx = amount of the unlabeled compound (ng)

Quantification of PCDFs and PCDDs in the sample extract is achieved by calculating the ratios of the mass spectral responses obtained for the ions characteristic of the labeled PCDFs/PCDDs to those of the appropriate internal standards, corrected for differences in response factor. The equation used for quantification was:

$$\text{Concentration (ng/g)} = (Ax.Qis)/Ais.W.RF)$$

where: W = weight of sample (g)
RF = response factor

Because of the labeled internal standard was added prior to sample extraction and analysis, and the internal standard was quantified at the same time as the native components, any losses of PCDD/PCDF incurred during the analysis were accounted for by the above approach.

Detection limits, i.e. the minimum detectable concentrations required to produce a signal 2.5 times the average background signal, were calculated for each PCDF/PCDD congener class. For each class, the average width of the baseline noise-band for the native compounds and the peak height for a known concentration of the associated ^{13}C-labeled analog were measured manually. Measurements of the noise band were made in a region of the selected ion current profile that was free of interferences and as close as practicable in the plot to the peak for the corresponding ^{13}C-labeled compound. The detection limit DL was calculated using the relationship:

$$DL = (2.5)(Ax.Qis)/(Ais.RF.W)$$

where: Ax = height of the noise band of the selected mass for the unlabeled compound
Ais = height of the peak corresponding to the labeled compound of the same congener class.

3. GC/MS ANALYSIS

Samples and standards were analyzed using combined capillary column gas chromatography/low-resolution mass spectrometry (HRGC/LRMS or GC/MS).

The gas chromatograph, a Varian 3700, was equipped with a 60 m x 0.25 mm i.d. fused-silica SP-2330 (Supelco) column. Samples were injected in the splitless mode, with the column programmed from 85C to 250C at a rate of 15C/min, and held at the upper limit for 60 minutes. The SP-2330 column has been shown to separate the 2,3,7,8-TCDD from all other tetra-isomers, and to partially separate the 2,3,7,8-TCDF from the 2,3,4,8-TCDF. However, it is unable to resolve 1,2,3,7,8-PnCDF from 1,2,3,4,8-PnCDF and 1,2,3,4,7,8-HxCDF from 1,2,3,4,7,9-HxCDF. These isomers can be separated to some extent on less polar columns, such as OV-17 or DB-5.

All data were acquired by software-controlled multiple ion detection using two ion masses from the molecular ion cluster for each of the PCDF and PCDD levels of chlorination. Two ions were also monitored for each internal standard. This permitted the calculation and comparison of the isotope ratios with their theoretical values to verify their identity. Several additional ion masses were monitored to indicate possible interferences from chlorinated diphenyl ethers. The presence of these compounds could give rise to fragment ions with the same masses as those monitored for the PCDFs. Therefore, the absence of a co-response for the diphenyl ether ion mass when a PCDF compound shows a positive response was taken to indicate that the signal for the PCDF was "real".

A listing of the masses that were monitored, along with the theoretical isotope ratios, is given in the Table. In order to obtain maximum sensitivity and selectivity during an experiment, the run was divided into four adjoining time windows. Each window contained up to 16 ion masses (for specific native and labeled PCDFs and PCDDs, and chlorinated diphenyl ethers), and the sample dwell times and interchannel delay times were chosen to give the most sensitive and rapid cycle time. Since the GC column used in this work did not yield complete separation of each chlorination level of PCDFs/PCDDs from the other groups in a given time window, some of the ion masses were included in more than one window to ensure that all isomers from a particular congener class were monitored.

The major criteria which were used for confirming the presence of specific PCDFs/PCDDs in the samples analyzed were the following: (1) correct retention times for each PCDF/PCDD of interest relative to the appropriate isotopically-labeled internal standard(s), (2) intensity ratio for $(M)^+/(M+2)^+$ within 10% of theoretically-expected ratio (see Table), and (3) signal-to-noise response for each PCDF/PCDD of interest greater than 2.5:1 for both ion masses monitored.

QUANTIFICATION

Ideally, each of the 210 separate isomers of PCDF and PCDD should be quantified using the instrument response of the corresponding labeled internal

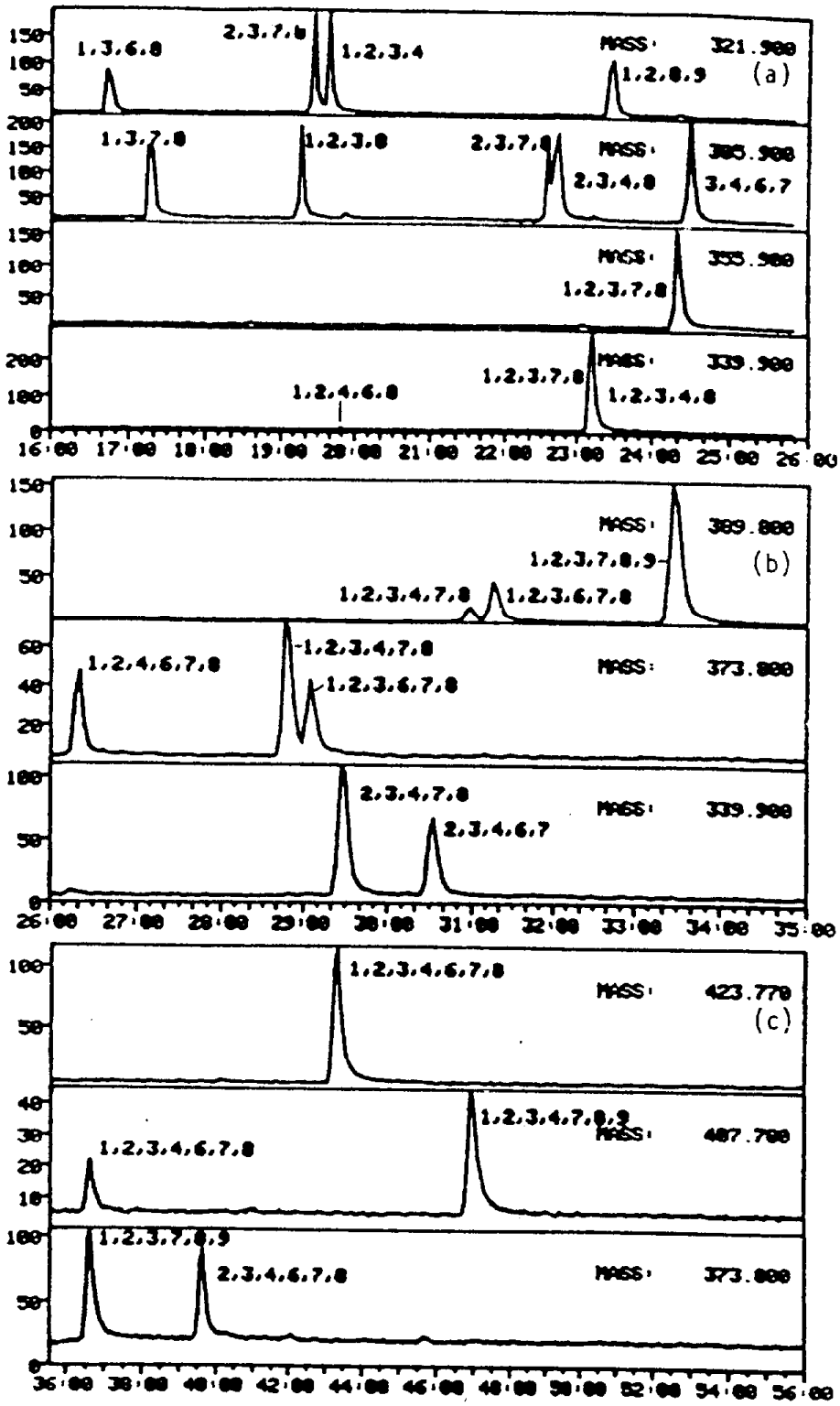

Figure. Selected ion current profiles of chromatography performance solution using SP-2330 capillary column.
(a) TCDD/TCDF/PnCDD/PnCDF components in 1st time window;
(b) HxCDD/HxCDF/PnCDF components in 2nd time window;
(c) HpCDD/HpCDF/HxCDF components in 3rd time window.

ANALYTICAL SEQUENCE IN GC/MS DETERMINATION OF PCDFs AND PCDDs

Time Window	Start (min)	Stop (min)	GC Column Temp (C)	Temp Prog Rate (C/min)	Cycle Time (ms)	Dwell Time (ms)	Compounds Monitored	Ions Monitored (m/z) Mass 1	Mass 2	Isotope Ratio [Mass 1/Mass 2]
1	14:00	26:00	85	15	3200	197	TCDF	303.9	305.9	0.77
							13C-TCDF	315.9	317.9	0.77
							TCDD	319.9	321.9	0.77
							13C-TCDD	331.9	333.9	0.77
							PnCDF	337.9	339.9	0.61
							13C-PnCDF	349.9	351.9	0.61
							PnCDD	355.9	357.9	1.54
							HxDPE*	373.8	---	---
							HpDPE*	407.8	---	---
2	26:00	35:30	250	---	3167	195	PnCDF	337.9	339.9	0.61
							PnCDF#	274.9	---	---
							PnCDD	355.9	357.9	1.54
							PnCDD#	290.9	---	---
							HxCDF	373.8	375.8	1.23
							13C-HxCDF	385.9	387.9	1.23
							HxCDF#	310.9	---	---
							HxCDD	389.8	391.8	1.23
							HxCDD#	326.9	---	---
							HpDPE*	407.8	---	---
							ODPE*	443.8	---	---
3	35:30	56:00	250	---	3750	372	HxCDF	373.8	375.8	1.23
							HxCDD	389.9	391.8	1.23
							HpCDF	407.8	409.8	1.03
							HpCDD	423.8	425.8	1.03
							ODPE*	443.8	---	---
							NDPE*	477.7	---	---
4	56:00	66:00	250	---	3750	534	OCDF	441.7	443.7	0.88
							OCDD	457.7	459.7	0.88
							13C-OCDD	469.8	471.8	0.88
							DDPE*	511.7	---	---

* HxDPE, HpDPE, ODPE, NDPE, DDPE designate hexa-, hepta-, octa-, nona-, and decachlorodiphenyl ethers, resp.

Mass monitored to identify fragment ion [M-COCl]

APPENDIX H

RESULTS OF STATE BOARD CDD AND CDF 2,3,7,8 CONGENER-SPECIFIC ANALYSES

TABLE H.1

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS, PHASE 1: SAWMILL A: COMPARISON OF ON-SITE DRUM OF COMMERCIAL PENTACHLOROPHENATE FORMULATION AND SLUDGE FROM DIP TANK

	COMMERCIAL Na-PCP		DIP TANK SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDDs (ppb)				
Total TetraCDD	1.9	<1	DL 2.2	<1
2,3,7,8	DL 1.4	<1	DL 2.7	<1
% 2,3,7,8 of total tetraCDD	-	-	-	-
Total PentaCDD	140	304	28	107
1,2,3,7,8	28.3	24	DL 15.9	10
% 2,3,7,8 of total pentaCDD	20%	13%	-	9%
Total HexaCDD	14,000	5,700	3,630	2,600
1,2,3,4,7,8	DL 6.1	ND	DL 12.5	ND
1,2,3,6,7,8	4,050	3,600	1,790	1,800
1,2,3,7,8,9	ND	ND	NR	ND
Total 2,3,7,8	4,050	3,600	1,790	1,800
% 2,3,7,8 of total hexaCDD	29%	63%	49%	69%
Total Hepta-CDD	100,000	40,000	36,400	42,000
1,2,3,4,6,7,8	33,800	28,000	15,400	25,000
% 2,3,7,8 of total heptaCDD	34%	70%	42%	60%
OctaCDD	81,000	13,000	115,000	155,000

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ ND is less than 50 ppb; <1 = reported as less than 1 ppb.

TABLE H.2

RESULTS OF CDD AND CDF CONGENER SPECIFIC ANALYSIS,
PHASE 1: SAWMILL B: COMPARISON OF WET AND DRY SLUDGES
SAMPLED FROM ABANDONED DIP TANK

	WET SLUDGE		DRY SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDDs (ppb)				
Total TetraCDD	DL 1.8	<1	8.4	60
2,3,7,8	DL 2.1	<1	8.3	11
% 2,3,7,8 of total tetraCDD	-	-	99%	18%
Total PentaCDD	25	246	720	1,298
1,2,3,7,8	34.3	14	185	212
% 2,3,7,8 of total pentaCDD	64%[3/]	5.7%	26%	16%
Total HexaCDD	640	400	6,200	8,600
1,2,3,4,7,8	DL 3.5	ND	93	ND
1,2,3,6,7,8	316	200	2,980	5,300
1,2,3,7,8,9	NR	ND	NR	ND
Total 2,3,7,8	316	200	3,073	5,300
% 2,3,7,8 of total hexaCDD	49%	50%	50%	62%
Total HeptaCDD	3,000	3,400	14,000	22,000
1,2,3,4,6,7,8	1,600	2,200	8,030	14,000
% 2,3,7,8 of total heptaCDD	53%	65%	57%	64%
OctaCDD	7,200	1,600	63,000	40,000

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ ND is less than 50 ppb; <1 = reported as less than 1 ppb.

3/ 64% based on DB-5 column; a previous analysis reported 19% of the total as 1,2,3,7,8-pentaCDD.

TABLE H.1 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS, PHASE 1: SAWMILL A: COMPARISON OF ON-SITE DRUM OF COMMERCIAL PENTACHLOROPHENATE FORMULATION AND SLUDGE FROM DIP TANK

	COMMERCIAL Na-PCP		DIP TANK SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDFs (ppb)				
Total TetraCDF	1,200	1,671	412	354
2,3,7,8	149	253	140	69
% 2,3,7,8 of total tetraCDF	12%	15%	34%	19%
Total PentaCDF	6,400	10,000	2,970	5,100
1,2,3,7,8	319	265	131	80
2,3,4,7,8	324	319	119	110
Total 2,3,7,8	643	584	250	190
% 2,3,7,8 of total pentaCDF	10%	6%	8%	4%
Total HexaCDF	49,000	7,200	8,700	8,500
1,2,3,4,7,8	DL 2.8	200	DL 8.5	300
1,2,3,6,7,8	225		145	
1,2,3,7,8,9	480	300	DL 14.3	300
2,3,4,6,7,8	DL 385	<100	DL 16.5	<100
Total 2,3,7,8	705	500	145	600
% 2,3,7,8 of total hexaCDF	1.4%	7%	2%	7%
Total HeptaCDF	91,000	9,700	9,300	8,000
1,2,3,4,6,7,8	6,190	3,900	2,270	3,000
1,2,3,4,7,8,9	154	ND	DL 100	<100
Total 2,3,7,8	6,344	3,900	2,270	3,000
% 2,3,7,8 of total heptaCDF	7%	40%	24%	38%
OctaCDF	36,000	1,000	3,890	1,800

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.0 = not detected at detection limit of 1.0 ppb.

2/ Sweden: 1,2,3,7,8- and 1,2,3,4,8-PentaCDF co-elute; 1,2,3,4,7,9-, 1,2,3,4,7,8- and 1,2,3,6,7,8-HexaCDF co-elute; ND is less than 50 ppb; <100 = reported as less than 100 ppb.

TABLE H.3

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS, PHASE 2: SAWMILL C: TETRACHLOROPHENATE

	TETRA-CHLOROPHENATE DRUM		DIP TANK LIQUID		DIP TANK SLUDGE		DIP TANK SLUDGE	
	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]
CDDS (ppb)								
Total TetraCDD	4.4	<1.7[3]	0.67	0.2	3.3	<3.0	8.3	6.5
2,3,7,8	<2.1	<1.7	<.10	<0.03	<1.5	<3.0	6.8	<1.7
% 2,3,7,8 of total tetraCDD	-	-	-	-	-	-	82%	-
Total PentaCDD	256	21	1.4	0.5	39.7	20	40.8	31
1,2,3,7,8	<5.1	<2.0	<.12	<0.03	<5.3	<1.7	<4.3	<0.9
% 2,3,7,8 of total pentaCDD	-	-	-	-	-	-	-	-
Total HexaCDD	1,630	851	15.5	7.6	410	397	532	421
1,2,3,4,7,8	<12.2	<4.7	8.2	<0.04	218	<3.0	<10.1	<2.6
1,2,3,6,7,8	667	323		3.3		192	242	192
1,2,3,7,8,9	NA[4]	14	NA	0.2	NA	7.9	NA	7
total 2,3,7,8	667	337	8.2	3.5	218	200	242	199
% 2,3,7,8 of total hexaCDD	41%	40%	53%	46%	53%	50%	45%	47%
Total HeptaCDD	1,360	805	25.7	14	1,380	1,564	1,720	1,444
1,2,3,4,6,7,8	849	527	15.3	9.3	797	974	976	898
% 2,3,7,8 of total heptaCDD	62%	65%	60%	66%	58%	62%	57%	62%
OctaCDD	1,450	5,680	106	289	1,290	9,770	4,960	11,056

FOOTNOTES

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331 column.

2/ IITRI - quantified on SP 2330.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

4/ NA - Not analyzed.

TABLE H.2 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 1: SAWMILL B: COMPARISON OF WET AND DRY SLUDGES
SAMPLED FROM ABANDONED DIP TANK

	WET SLUDGE		DRY SLUDGE	
	CAL[1/]	SWEDEN[2/]	CAL[1/]	SWEDEN[2/]
CDFs (ppb)				
Total TetraCDF	76	79	1,700	2,294
2,3,7,8	20	13	112	78
% 2,3,7,8 of total tetraCDF	26%	16%	7%	3%
Total PentaCDF	650	500	9,100	13,000
1,2,3,7,8	24	25	140	119
2,3,4,7,8	19	31	131	169
Total 2,3,7,8	43	56	271	288
% 2,3,7,8 of total pentaCDF	7%	11%	3%	2%
Total HexaCDF	1,530	900	6,600	9,200
1,2,3,4,7,8	DL 3.9	ND	DL 14	100
1,2,3,6,7,8	19		104	
1,2,3,7,8,9	DL 11.3	ND	DL 23.5	200
2,3,4,6,7,8	26	ND	177	ND
Total 2,3,7,8	45	-	281	300
% 2,3,7,8 of total hexaCDF	3%	0	4%	3%
Total HeptaCDF	960	700	3,000	4,100
1,2,3,4,6,7,8	446	300	1,120	1,900
1,2,3,4,7,8,9	DL 17	ND	DL 32.5	ND
Total 2,3,7,8	446	300	1,120	1,900
% 2,3,7,8 of total heptaCDF	46%	43%	37%	46%
OctaCDF	270	100	2,400	600

FOOTNOTES

1/ Isomer group totals quantified on DB-5 column; specific 2,3,7,8 congeners quantified on Supelco 2331 column; NR = not reported; DL 1.4 = not detected at detection limit of 1.4 ppb.

2/ Sweden: 1,2,3,7,8- and 1,2,3,4,8-PentaCDF co-elute; 1,2,3,4,7,9-, 1,2,3,4,7,8- and 1,2,3,6,7,8-HexaCDF co-elute; ND is less than 50 ppb; <100 = reported as less than 100 ppb.

TABLE H.4

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: WOOD TREATMENT PLANT

	"BLOOM"		COMMERCIAL"		SOIL (MOUTH OF RETORT)		SUMP (LIQUID)	
CDDs (ppb)	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]
Total TetraCDD	<2.0[3]	<1.8	<.32	<2.3	<.48	<1.1	<1.4	5.5
2,3,7,8	<3.0	<1.8	<2.7	<2.3	<2.2	<1.1	<4.7	<0.7
% 2,3,7,8 of total tetraCDD	-	-	-	-	-	-	-	-
Total PentaCDD	109	30	<1.3	<3.5	4.4	<1.2	39	<1.4
1,2,3,7,8	90	21	<14.3	<3.5	<12.8	<1.2	<19.7	<1.4
% 2,3,7,8 of total pentaCDD	82%	70%	-	-	-	-	-	-
Total HexaCDD	2,020	1,140	127	144	215	271	1,420	84
1,2,3,4,7,8	145	103	<8.4	<16.6	<12.3	9.4	30	12
1,2,3,6,7,8	510	347	65	<16.6	70	90	384	28
1,2,3,7,8,9	NA[4]	8	NA[4]	<16.6	NA[4]	22	NA[4]	14
TOTAL 2,3,7,8	655	458	65	-	70	121	414	54
% 2,3,7,8 of total HexaCDD	32%	40%	51%	-	33%	45%	29%	64%
Total HeptaCDD	31,800	21,552	8,490	10,568	1,940	2,890	12,900	548
1,2,3,4,6,7,8	30,300	16,837	5,980	7,319	1,204	1,839	8,270	343
% 2,3,7,8 of total HeptaCDD	95%	78%	70%	69%	62%	64%	64%	63%
OctaCDD	135,000	145,693	115,000	143,043	8,040	8,397	77,090	10,707

FOOTNOTES

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331.

2/ IITRI - quantified on SP 2330.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

4/ NA - Not analyzed.

TABLE H.3 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: SAWMILL C: TETRACHLOROPHENATE

	TETRA-CHLOROPHENATE DRUM		DIP TANK LIQUID		DIP TANK SLUDGE		DIP TANK SLUDGE	
	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]	CAL[1]	IITRI[2]
CDFs (ppb)								
Total TetraCDF	1,280	1,179	10.5	5.3	234	534	279	522
2,3,7,8	262	138	2.8	1.2	46	62	65	64
% 2,3,7,8 of total tetraCD	20%	12%	27%	23%	20%	12%	23%	12%
Total PentaCDF	7,190	1,765	53	13	1,150	658	1,310	555
1,2,3,7,8 & 1,2,3,4,8	262	<2.0[3]	2.1	1.5	50	95	54	82
2,3,4,7,8	88	59	1.3	0.6	28	60	26	50
total 2,3,7,8	350	59	3.4	2.1	77	155	80	132
% 2,3,7,8 of total pentaCDF	5%	3%	6%	16%	7%	24%	6%	24%
Total HexaCDF	6,240	4,657	58	36	1,470	1,806	1,850	2,227
1,2,3,4,7,8 & 1,2,3,4,7,9	20	<4.7	0.09	0.2	16	18	17	23
1,2,3,6,7,8	<12.5	<4.7	22	0.1		7.6	6	9.2
1,2,3,7,8,9	46	<4.7	<.23	<0.04	<11.1	<3.0	31	<2.6
2,3,4,6,7,8	<14.6	30	0.94	0.4	<7.0	28	<7.5	36
total 2,3,7,8	66	30	23	0.7	16	54	54	68
% 2,3,7,8 of total hexaCDF	1%	0.6%	40%	2%	1%	3%	3%	3%
Total HeptaCDF	2,750	1,338	32	14	963	921	1,180	774
1,2,3,4,6,7,8	808	582	11	6.3	306	402	342	341
1,2,3,4,7,8,9	<18.7	<3.7	<.36	<0.03	<10.6	519	<7.1	<1.9
total 2,3,7,8	808	582	11	6.3	306	921	342	341
%2,3,7,8 of total heptaCDF	29%	43%	34%	45%	32%	100%	29%	44%
OctaCDF	132	<337	6.3	1.1	378	102	375	100

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331 column.

2/ IITRI - SP 2330 column - note co-eluting pentaCDF and hexaCDF congener.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

TABLE H.5

CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSES: PERCENT SPECIFIC 2,3,7,8 ISOMERS DETECTED IN EACH CDD AND CDF ISOMER GROUP. PHASE 1: SAWMILLS A AND B

	COMMERCIAL Na-PCP FORMULATION	SAWMILL A SLUDGE	SAWMILL B LIQUID SLUDGE	SAWMILL B DRY SLUDGE
CDDs				
2,3,7,8 tetraCDD[1]	ND	ND	ND	18% 99%[3]
1,2,3,7,8 pentaCDD[2]	12%	ND 9%[3]	5.7% 38%[3]	20%
1,2,3,4,7,8 hexaCDD[2]	ND	ND	ND	ND 1.5%[3]
1,2,3,6,7,8 hexaCDD	39%	58%	50%	56%
1,2,3,7,8,9 hexaCDD	ND	ND	ND	ND
1,2,3,4,6,7,8 heptaCDD[2]	44%	60%	64%	61%
CDFs				
2,3,7,8 tetraCDF[1]	14%	27%	22%	4.8%
1,2,3,7,8 pentaCDF	3.6%	2.6%	4.3%	1.2%
2,3,4,7,8 pentaCDF	3.9%	2.8%	4.3%	1.4%
1,2,3,4,7,8 hexaCDF	ND	ND	ND	ND
1,2,3,6,7,8 hexaCDF	0.8%	2.6%	ND 1.2%[3]	1.3%
1,2,3,7,8,9 hexaCDF	1.3%	ND 3.5%[3]	ND	ND 2.2%[3]
2,3,4,6,7,8 hexaCDF	ND	ND	ND 1.7%[3]	ND 2.7%[3]
1,2,3,4,6,7,8 heptaCDF	10%	30%	45%	43%
1,2,3,4,7,8,9 heptaCDF	<0.2%	ND	ND	ND

FOOTNOTES

ND = None Detected
1/ Internal standard used by both labs (also used OctaCDD).
2/ Internal standard used by Cal Labs.
3/ Results of both labs given.

TABLE H.4 (continued)

RESULTS OF CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSIS,
PHASE 2: WOOD TREATMENT PLANT

CDFs (ppb)	"BLOOM" CAL[1]	"BLOOM" IITRI[2]	COMMERCIAL" CAL[1]	COMMERCIAL" IITRI[2]	SOIL (MOUTH OF RETORT) CAL[1]	SOIL (MOUTH OF RETORT) IITRI[2]	SUMP (LIQUID) CAL[1]	SUMP (LIQUID) IITRI[2]
Total TetraCDF	18	43	<0.2[3]	1.3	0.67	4.9	9.1	26
2,3,7,8	4.4	4.4	<3.6	<1.9	<4.7	<1.3	<5.2	<1.3
% 2,3,7,8 of total tetraCDF	24%	10%	-	-	-	-	-	-
Total PentaCDF	561	203	<2.9	<3.5	72.6	12	484	27
1,2,3,7,8 & 1,2,3,4,8	53	<2.2	<9.1	<3.5	<7.2	4.8	32	8.4
2,3,4,7,8	31	27	<9.1	<3.5	<5.5	<1.2	18	4.2
total 2,3,7,8	84	27	-	-	-	4.8	50	13
% 2,3,7,8 of total pentaCDF	15%	13%	-	-	-	40%	10%	47%
Total HexaCDF	4,520	3,637	251	209	211	219	2,440	168
1,2,3,4,7,8 & 1,2,3,4,7,9	902	662	12.8	<16.6	6.0	<5.0	61	<3.6
1,2,3,6,7,8	236	232	<12.1	<16.6	<11.8	<5.0	25	<3.6
1,2,3,7,8,9	94	<3.9	<14.4	<16.6	<17.8	<5.0	50	<3.6
2,3,4,6,7,8	<8.7	85	<14.3	37	<13.4	17	<20.6	<3.6
total 2,3,7,8	1,232	979	13	37	6.0	17	136	-
% 2,3,7,8 of total hexaCDF	27%	27%	5%	18%	3%	8%	6%	-
Total HeptaCDF	17,400	8,606	4,240	2,613	458	388	2,590	111
1,2,3,4,6,7,8	11,600	5,056	747	425	747	145	900	40
1,2,3,4,7,8,9	1,490	396	110	120	110	12	<50	16
total 2,3,7,8	13,090	5,452	857	545	847	157	900	56
%2,3,7,8 of total heptaCDF	75%	63%	20%	21%	185%	40%	35%	50%
OctaCDF	223,000	12,323	175,000	11,648	1,470	222	3,600	<326

FOOTNOTES

1/ Cal Labs - totals quantified on DB-5 column; Congener specific quantified on SP 2331 column.

2/ IITRI - quantified on SP 2330.

3/ <1.0 indicates not detected at a detection limit of 1.0 ppb.

TABLE H.5 (continued)

CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSES:
PERCENT SPECIFIC 2,3,7,8 ISOMERS DETECTED IN EACH
ISOMER GROUP. PHASE 2: WOOD TREATMENT PLANT: COMMERCIAL PCP

	"BLOOM"	"COMMERCIAL"	SOIL AT RETORT MOUTH	SUMP LIQUID
CDDs				
2,3,7,8 tetraCDD[1]	ND	ND	ND	ND
1,2,3,7,8 pentaCDD[2]	80%	ND	ND	ND
1,2,3,4,7,8 hexaCDD[2]	7.8%	ND	ND 3.5%[4]	2.1% 14%[4]
1,2,3,6,7,8 hexaCDD	27%	51%	33%	27% 33%[4]
1,2,3,7,8,9 hexaCDD	NA 0.7%[4]	NA ND[4]	NA 8.1%[4]	NA 17%[4]
1,2,3,4,6,7,8 heptaCDD	88%	70%	63%	64%
CDFs				
2,3,7,8 tetraCDF[1]	14%	ND	ND	ND
1,2,3,7,8 pentaCDF[3]	9.4% ND[4]	ND	ND 40%[4]	6.6% 31%[4]
2,3,4,7,8 pentaCDF	5.5% 13%[4]	ND	ND	3.7% 16%[4]
1,2,3,4,7,8 hexaCDF[3]	19%	5.1% ND[4]	2.8% ND[4]	2.5% ND[4]
1,2,3,6,7,8 hexaCDF	5.7%	ND	ND	1% ND[4]
1,2,3,7,8,9 hexaCDF	2.1% ND[4]	ND	ND	2% ND[4]
2,3,4,6,7,8 hexaCDF	ND 2.3%[4]	ND 18%[4]	ND 7.8%[4]	ND
1,2,3,4,6,7,8 heptaCDF	64%	17%	163% 37%[4]	35%
1,2,3,4,7,8,9 heptaCDF	7.3%	3.1%	24% 3.1%[4]	ND 14%[4]

FOOTNOTES

ND = None Detected
NA = Not Analyzed

1/ Internal standard used by both labs (also used OctaCDD).
2/ Internal standard used by Cal Labs.
3/ Internal standard used by IITRI.
4/ Results of both labs given; first value is Cal Labs, second is IITRI.

TABLE H.5 (continued)

CDD AND CDF 2,3,7,8 CONGENER SPECIFIC ANALYSES:
PERCENT SPECIFIC 2,3,7,8 ISOMERS DETECTED IN EACH
ISOMER GROUP. PHASE 2: SAWMILL C, TETRACHLOROPHENATE

	COMMERCIAL TETRACHLORO-PHENATE FORMULATION	DIP TANK LIQUID	DIP TANK SLUDGE	DIP TANK SLUDGE
CDDs				
2,3,7,8 tetraCDD[1/]	ND	ND	ND	82% ND[4/]
1,2,3,7,8 pentaCDD[2/]	ND	ND	ND	ND
1,2,3,4,7,8 hexaCDD[2/]	ND	ND	ND	ND
1,2,3,6,7,8 hexaCDD	40%	50%	51%	54%
1,2,3,7,8,9 hexaCDD	NA 1.6%[4/]	NA 2.6%[4/]	NA[4/] 2.0%	NA[4/] 1.7%
1,2,3,4,6,7,8 heptaCDD[2/]	64%	60%	60%	59%
CDFs				
2,3,7,8 tetraCDF[1/]	16%	24%	14%	16%
1,2,3,7,8 pentaCDF[3/]	3.6% ND[4/]	5.5%	8%	8.4%
2,3,4,7,8 pentaCDF	1.6%	2.9%	4.7%	4.1%
1,2,3,4,7,8 hexaCDF[3/]	0.3% ND[4/]	3.1%	1%	1%
1,2,3,6,7,8 hexaCDF	ND	24%	0.4%	0.7%
1,2,3,7,8,9 hexaCDF	0.7% ND[4/]	ND	ND	1.7% ND[4/]
2,3,4,6,7,8 hexaCDF	ND 0.6%[4/]	1.4%	ND 1.6%	ND[4/] 1.6%[4/]
1,2,3,4,6,7,8 heptaCDF	34%	38%	38%	35%
1,2,3,4,7,8,9 heptaCDF	ND	ND	ND 56%[4/]	ND

FOOTNOTES

ND = None Detected
NA = Not Analyzed

1/ Internal standard used by both labs (also used OctaCDD).
2/ Internal standard used by Cal Labs.
3/ Internal standard used by IITRI.
4/ Results of both labs given; first value is Cal Labs, second is IITRI.

APPENDIX J

BACKGROUND DOCUMENTS FOR CHAPTER 8: HAZARD EVALUATION

PAGE

BACKGROUND DOCUMENTS:

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

November 4, 1986

SAB-EC-87-008

Honorable Lee M. Thomas
Administrator
U. S. Environmental
Protection Agency
401 M Street, S. W.
Washington, D. C. 20460

OFFICE OF
THE ADMINISTRATOR

Dear Mr. Thomas:

The Science Advisory Board's (SAB) Dioxin Toxic Equivalency Methodology Subcommittee met in public session on September 8 to review a draft document prepared by the Agency's Risk Assessment Forum and entitled "Interim Procedures for Estimating Risk Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CDDs and CDFs)." The document sets forth an approach for assessing the hazard of CDD and CDF mixtures relative to the toxicity of the 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD) isomer.

The request for SAB review of this Toxic Equivalency Factor (TEF) methodology originated on February 27, 1986 from Assistant Administrator for Air, Craig Potter, who recommended SAB review in relation to his office's ongoing interest in assessing dioxin risks associated with municipal waste combustion. The SAB Executive Committee accepted this request and formed the Dioxin Toxic Equivalency Methodology Subcommittee to carry out the review. The Subcommittee approached its task with the assumption that it was reviewing a generic methodology as opposed to one that was limited solely to the issue of municipal waste combustion.

The Subcommittee's report consists of two sections: 1) its consensus statement on the draft document in its current form, and 2) comments on individual steps that EPA should initiate to improve the document or the scientific data base for toxic equivalency evaluation. The Subcommittee has already forwarded the comments of its individual members to the Agency's staff.

The Subcommittee generally concludes that the draft document represents a successful interim attempt to articulate a scientific rationale and procedures for developing risk management decisions for mixtures which contain CDDs and CDFs related in structure and activity to TCDD. The Subcommittee's major recommendations include: placing greater emphasis on

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Report of the Science Advisory Board's Dioxin Toxic Equivalency Methodology Subcommittee Following Its Evaluation of EPA's Toxic Equivalency Factor Methodology for CDDs and CDFs

A. Major Subcommittee Conclusions

EPA has proposed interim procedures for estimating health risks for CDDs and CDFs based on the premises that: (a) toxicity equivalence factors can be assigned to untested (or incompletely tested) compounds on the basis of structure/activity relationships; and (b) the toxicity of mixtures of these compounds can be approximated for policy purposes by the sums of their TEF times concentrations. Empirically, the present proposal falls generally between the positions adopted by certain European countries, which rank 2,3,7,8 TCDD far above any other congener in toxicity, and that initially proposed by the state of California, which equates all the dioxin congeners. All have used similar scientific assumptions in developing policy.

The Subcommittee agrees that the congeners of CDDs and CDFs constitute a class of chemical substances that share similar structural relationships and qualitatively similar toxic effects and, therefore, can reasonably be considered together. From the limited toxicologic data available it seems reasonable, too, to consider those tetra-to hexa-chlorinated compounds with chlorine substitutions at the lateral 2,3,7,8 positions as a closely related subclass in terms of biological activity and environmental fate.

The Subcommittee also concurs that the problems in assessing the health risks of dibenzo-p-dioxins and dibenzofurans are two-fold. They include: limited information from human or experimental studies about the hazards from exposure to these compounds (few of the 75 CDDs and 135 CDFs have been tested at all) and even more limited information about their possible interactions in mixtures. Indications of interactions, mostly additive, are found in certain experimental model systems (e.g. binary combinations). Not addressed in the draft document, however, is the possibility of chemical and toxicologic

toxicokinetics, including metabolism; assigning priority to using human data, when available; validating the TEF methodology by selected testing of hypotheses; articulating clearly the decision steps, assumptions and methods of calculation; restating and re-emphasizing the interim nature of the methodology; and addressing the possibility of chemical and toxicological interactions with other types of compounds in complex environmental mixtures.

We appreciate the opportunity to review the TEF methodology and to present our technical evaluation. We request that the Agency formally respond to the scientific advice provided in the attached report.

Sincerely,

Richard Griesemer

Richard Griesemer, Chairman
Dioxin Toxic Equivalency
Methodology Subcommittee

Norton Nelson, Chairman
Executive Committee

However, EPA should not abandon its exploration of other approaches to estimating risks for substances in mixtures. For example, where variability in the composition of environmental samples is not wide, a reference standard approach might be used (similar to those used in toxicology for selecting a reference cigarette or a representative blend of gasolines). As another example, the incorporation of a small amount of radiolabeled test compound into a representative and defined mixture might be one useful way of determining in vivo whether the uptake and metabolism of one congener is greatly modified by the presence of other substances in a mixture.

Some additional technical comments that the Subcommittee wishes to draw to the Agency's attention include: 1) perceptions by many Subcommittee members of an over-reliance upon the postulated mechanisms of the Ah receptor/AHH enzyme induction upon which to gauge relative and absolute toxicity; 2) the need to discuss the work of Matsumura, Rozman, Greenlee, Poellinger and others on additional toxicologically significant effects of the dioxins other than those associated with receptor binding or with cytochrome P-450 induction; 3) observations of a disassociation between AHH induction and cytotoxicity in studies on the gonado toxicity of TCDD; and 4) examination of the extent to which the longer biological half-life of higher chlorinated dioxin isomers, as compared to 2,3,7,8-TCDD, counter-balances their lesser in vivo potency.

B. Major Subcommittee Recommendations

The Subcommittee has several recommendations for improving the report. First, the draft report narrative is relatively brief and may not be readily understood by those not familiar with dioxins. For example, four

interactions with other types of compounds in complex environmental mixtures, especially solvents that might affect uptake and retention by the body. EPA should address the latter subject in the TEF document, perhaps with more specific reference to its recently published Risk Assessment Guidelines and to three National Academy of Sciences' reviews on toxicological interactions, the last of which is currently being prepared for EPA and the National Institute of Environmental Health Sciences. The Subcommittee also questions the basis for including or excluding other chemicals with effects similar to CDDs and CDFs, such as chlorinated biphenylenes.

Based upon its review of the draft document, the Subcommittee concludes that the method proposed by EPA is a reasonable interim approach to assessing the health risks associated with exposure to mixtures of CDDs and CDFs for risk management purposes. It is necessary, however, as lessons are learned from toxicologic research and from application, the approach should be re-evaluated systematically by EPA. Moreover, attempts should be made to validate the method by selected experimental testing of hypotheses. For example, more data are needed on in vivo potencies of additional PCDDs and PCDFs to compare with in vitro test results. The assumption of additivity can be evaluated by comparing observed activities with predicted activities in selected tests.

The Subcommittee recommends that EPA place more emphasis on the interim nature of the method in the document. The Subcommittee anticipates that, over time, the method will be modified and eventually superseded as more precise data become available. Meanwhile, the general method proposed appears to have utility for this and for other classes of closely related compounds where toxicologic data are incomplete. Application of structure activity relationships is an old and established practice of demonstrated usefulness in pharmacology and toxicology.

toxicologic practice of evaluating all endpoints, and selecting the ones most reliable, sensitive, and important for risk assessment. Thus, columns should be added to the tables in the document for other important toxic endpoints including immunotoxicity, thymic atrophy, body weight, and enzyme induction in vivo. The limited data points from which TEFs are currently derived (e.g. carcinogenicity of 2,3,7,8-TCDD, 2,3,7,8-Hx CDDs and reproductive effects of those compounds plus 2,3,7,8-TCDF) should be critically re-examined and the range of experimental data and estimated potencies from all studies tabulated. The Subcommittee also recommends that EPA consider assigning higher relative TEFs to CDFs in general, and 2,3,4,7,8-PeCDF in particular.

The Subcommittee strongly believes that EPA should assign greater priority to obtaining and using data on toxicokinetics, including metabolism. The rates of uptake and distribution of compounds alone and in mixtures are important measures of bioavailability and dosimetry. The kinetics of metabolism and excretion, along with those of receptor kinetics and affinities, should be especially useful for interspecies comparisons and for estimating risks for this particular class of compounds.

The Subcommittee wishes to emphasize that the method proposed may lack scientific validity. The associated errors have not been quantified. It is important, therefore, that the Agency make every effort to validate the method. The Subcommittee recommends periodic review and analysis as better data are obtained, and that EPA make systematic efforts to obtain critically important data, including that from in vivo tests on compounds

possible approaches are introduced and one (TEF) selected, but the document does not clarify what the other three aproaches are and the reasons for their rejection. The first approach, long-term animal testing, might be appropriate for municipal incinerator fly ash, where analytic data suggest there is a characteristic pattern of composition. The second approach (short-term assays) is not clearly described (not even whether they are in vivo or in vitro). The third approach, additivity of the toxicity of components, is at first rejected in the narrative but then forms the basis for handling the equivalents to 2,3,7,8-TCDD in mixtures.

Because the draft document presents a procedure, it is essential that the decision steps be clearly articulated, the assumptions made explicit, and the mechanics of calculating be illustrated in a stepwise fashion. To approach the subject from the viewpoint of studying the whole class of pollutants and to avoid bias by selecting data, the Subcommittee recommends that the tabular data be enlarged to include all compounds with zero to eight substituted chlorines. Biological activity has been reported for di- and tri-CDDs, and carcinogenicity studies exist for DD and 2,7 DCDD, as examples. Moreover, the activity of brominated and other substituted compounds should also be indicated and a specific effort encouraged to collect data on non-chlorine substituted compounds.

In contrast with the document's first priority on carcinogenic and then on teratologic effects in animals, the Subcommittee recommends that the TEF methodology assign first priority to human data when it exists. In evaluating experimental data, EPA should continue to follow the current

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with representative positional substitutions. Efforts should continue to develop methods for assaying the biologic activity of important mixtures (e.g. fly ash) in in vitro systems, using other cells in addition to hepatocytes and other endpoints in addition to AHH activity. Until the uncertainties are reduced, the interim TEF method should be largely reserved for specific situations where the components of the mixture are known, where the composition of the mixture is not expected to vary much with time, and where the extrapolations are consistent with existing animal data.

CALIFORNIA SITE MITIGATION DECISION TREE MANUAL

EXECUTIVE SUMMARY

Introduction

The purpose of the California Site Mitigation Decision Tree is to provide State decision makers with a standardized approach to setting site-specific mitigation criteria. The Decision Tree will also facilitate evaluation of remedial action alternatives to select the best plan based on scientific considerations of factors relating to public health and the environment while considering demographic factors, local concerns, and other variables.

Major elements contained within the Decision Tree include processes for setting Applied Action Levels (AALs) for contaminants in soil, water, air and biota in an expeditious manner; identifying specific data to be gathered; identifying preferred data gathering techniques and developing site mitigation criteria for alternative remedial actions.

It should be noted that the Decision Tree process establishes both Applied Action Levels (AALs) and site mitigation criteria. AALs are exposure criteria applied to all sites throughout the State. AALs delineate concentrations of toxic substances that, when exceeded, place a specified biological receptor at significant risk. Because AALs are biological receptor specific, not site specific, they have statewide applicability. The mitigation criteria, on the other hand, are site-specific criteria that a remedial action must achieve to keep the exposure level at the biological receptor below the AAL (i.e., below significant risk).

The Decision Tree process consists of five components. These components include Preliminary Site Appraisal, Site Assessment, Risk Appraisal, Environmental Fate and Risk Determination, and Determination of Mitigation Strategy and Remedial Action Plan Selection. The relationship between these components and an overview of the Decision Tree process are shown in Figure 1.

The Decision Tree process was designed to be applied to a variety of sites that may range from small, relatively simple sites to large, highly complex and difficult sites. Because of this diversity, the Decision Tree document is rather massive. However, its size should not intimidate the user. If the site is small and relatively easy to mitigate, many of the decision branches are never opened and the decision process is very rapid. If the site is complex, many of the decision branches must be opened and pursued. In either case, the decision points and the data requirements needed to make sound decisions are defined.

COMPONENT I: Preliminary Site Appraisal

Sites in California contaminated with hazardous wastes have been identified by regulatory agencies such as the State of California Department of Health Services, the California State Water Resources Control Board and the nine Regional Water Quality Control Boards, the United States Environmental Protection Agency, and a myriad of local agencies responsible property owners reporting contamination problems; and concerned citizens, including residents and past and present employees of companies that have contaminated sites. Preliminary Site Appraisal (PSA) is initiated by the discovery of a site which is potentially contaminated with hazardous substances. Based on the

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COMPONENT II: Site Assessment

The second component of the Decision Tree includes a preliminary evaluation of the site specific factors that affect the tendency of a hazardous substance to move between environmental compartments (air, soil, water, and biota). First, critical exposure pathways are identified. These pathways are the means by which exposure to contaminants in air, soil, water and biota occurs. Water pathways include both surface and ground waters.

In the first phase of identifying critical exposure pathways, determination of the current contaminant concentrations at points of exposure to biological receptors of concern should be made. The measurements or estimates made at this point are not thought of as static; concentrations change with time. Hazardous substances are not assumed to be isolated from biological receptors in separate environmental compartments. The Decision Tree process identifies data to be gathered for each pathway and cites preferred methods of data collection.

Typically, in this initial stage, samples are taken of waste, surface soils and shallow soils, runoff and surface water and ground water from existing wells. Sampling data from these site assessment activities will be the basis for a decision as to whether expanded air, soils, and ground water investigations will be necessary.

Expanded Assessment

If an expanded assessment of the site is warranted, evaluation of the environmental setting as it affects the behavior of the contaminants is required. The quantification of the meteorological, biological, soils and hydrologic systems at the site, together with information about the chemical and physical properties of the contaminants, forms a basis to evaluate the environmental fate of contaminants.

The collection of the necessary and sufficient data to adequately characterize the site and contaminants is the principal objective of this component. Site investigation programs are iterative in nature and will often require subsequent sampling and monitoring device installation to resolve issues that arise after data collected during initial site appraisal are analyzed. Although environmental compartments are presented as separate modules, contaminant transfer occurs across compartment boundaries and processes in one compartment often influence processes in another.

Quality Assurance/Quality Control

As the process is described in the Decision Tree Manual, data obtained from field sampling and analysis and/or literature values may be used to determine existing and future concentrations of chemical contaminants in environmental media (i.e., air, soil, water, and biota). Regardless of the source, it is important that these data be accurate, precise, complete, representative and comparable to other appropriate data.

characteristics of the wastes present and the features of the site itself, the site may be determined to be sufficiently hazardous to be placed on either the National Priority List (NPL) and/or the State Bond Act Expenditure Plan (CSBAEP).

The process to rank sites on the NPL and CSBAEP is primarily based on qualitative information, and does not include the detailed investigation required to fully characterize a site. Therefore, additional information may be required to implement subsequent components of the Decision Tree process. The Decision Tree process may also be used for sites that are not listed on the NPL and CSBAEP.

The PSA component of the Decision Tree identifies the universe of contaminants potentially present at the site using information acquired from the NPL or CSBAEP process. Additional investigation may be needed regarding the use of chemicals, production of wastes, and disposal practices at the site. The universe of contaminants is verified by a limited sampling program of the areas where contamination is most likely to be found.

An initial investigation of the adverse effects of the contaminants is necessary to determine the potential endangerment to human health. Determination of the likelihood of adverse effects occurring upon exposure to the contaminants is based upon available toxicologic data. Available data may show that adverse human health effects are imminent, and appropriate emergency action is necessary to protect public health.

Determination of the occurrence of health effects due to low level, chronic exposure is more difficult and requires additional investigation of the extent and magnitude of contamination, and the toxicologic properties of the contaminants, individually and collectively. Once it has been established that additional site assessment is necessary to provide data to develop appropriate remedial measures, the remaining components of the Decision Tree are initiated.

approaches are factored into the criteria to ensure a margin of protection for the biological receptors of concern.

The total exposure of biological receptors to toxic substances via various media is evaluated at their site(s) of exposure. The fractions of the AAL present in each media of exposure are added. When the total cumulative exposure exceeds an MEL, a significant risk to a biological receptor is indicated and a risk management process is warranted.

Exposure to substances that produce the same toxic manifestation or are considered likely to interact is also appraised. When the total cumulative exposures to toxic chemicals in all media of exposure constitute a significant risk to a biological receptor, the initiation of a risk management process is warranted.

Whenever an Applied Action Level is exceeded in any media of exposure, an assessment will be made to determine the necessity for interim actions to be implemented immediately to protect public health and the environment. A few examples of immediate interim actions are: fencing the contaminated site, covering exposed contaminated soils, and restricting use of water. Immediate interim actions will usually not be the ultimate containment or treatment strategy. Interim actions are developed primarily to reduce public exposure prior to initiating the final Remedial Action Plan (RAP).

COMPONENT IV: Environmental Fate and Risk Determination

Environmental Fate - Subsurface Conditions, Soils and Groundwater

In producing this section of the Decision Tree Manual, it is recognized that diverse subsurface conditions are encountered in hazardous waste site investigations and that considerable flexibility and professional judgement are often required to conduct an investigation of subsurface geology, hydrology, and soil and ground water contamination. Items to be considered include those factors that could act to transfer contaminants adsorbed to soil particles through the soil column. Factors of concern are: infiltration of precipitation, leakage of liquids from underground storage or conveyance structures, and spillage or other discharges to ground that could encourage leaching of contaminants from soil. In many cases, both current and future land use must be considered to evaluate the effect of environmental factors on the contaminants residing in the soil column.

Patterns of soil contamination existing at a hazardous waste site may often be the result of waste disposal events that took place over many years. Variations in the waste type, climate and precipitation, micro-structure of the soils, biological activity, and soil-chemical interactions can act to result in a complex pattern of soil contamination. Evaluation of patterns of soil contamination at a hazardous waste site should begin with recognition of any qualitative similarities or discernible trends that might be corrected with stratigraphy. Initial perceptions should be validated by actual field sampling.

The ground water investigation, including water quality and hydrological assessment, should occur in coordination with the subsurface soils investigation.

To ensure that all data used in the process described in this manual are representative of environmental conditions, the Quality Assurance/Quality Control Plans (QA/QC) used in the data generation need to be evaluated. The main components of a QA/QC plan that need special scrutiny are the basis for measurement, experimental information, statistical information (e.g., means, ranges, and standard deviation), and corroborative information.

The QA/QC plans for sampling and for analysis should be developed together. At a minimum, the data generators and users should work together in developing an integrated site-specific QA/QC plan.

COMPONENT III: Risk Appraisal

An evaluation of the effects produced by toxic substances which originate from waste sites centers on appraising the adverse impacts of these substances on the public health and the surrounding ecosystem. Every potential effect is not, and should not be, delineated by the appraisal process. Given the limited resources that are available and the complexity of the numerous sites scattered throughout California, the appraisal must focus on those biological receptors of concern that are potentially at risk. The Decision Tree process is aimed at ensuring their protection.

Three types of information are essential to evaluate the sites.

1. The toxic substances are identified from data collected in the Site Assessment Process.

2. The biological receptors of concern in the ecosystem potentially impacted by the toxic substances are identified in the Site Assessment Process.

3. The critical exposure pathways are delineated in the Site Assessment Process.

A criterion will be identified or developed for maximum acceptable exposure for toxic contaminants. The criteria are employed to identify significant adverse effects of the toxic contaminants on the biological receptors. These criteria, denoted as Applied Action Levels, are applicable statewide. An Applied Action Level (AAL) is specific to a toxic substance, a biological receptor and a medium of exposure.

The methodology employed to develop AALs is quite conventional. It is a compilation of the approaches outlined by the U.S. Environmental Protection Agency, the National Academy of Sciences and the California Department of Health Services. Toxic Substances are grouped into two catagories for the purpose of developing AALs. For carcinogens, mutagens and genotoxic teratogens no threshold for an adverse effect is assumed. The AALs are based on a maximum exposure level (MEL) which produces one adverse effect in a population of one million exposed. The MELs are determined from epidemiological research or long-term animal bioassays.

For other toxic agents a threshold for an adverse effect is assumed. The AAL is established, with a margin of safety, at the maximum exposure level which does not produce an adverse effect. Uncertainties associated with the above

Constant. Vapor pressure, defined as the pressure exerted by a gas when in equilibrium with the liquid or solid phase, is a useful screening indicator of the potential of a chemical to volatilize from land. The Henry's Law Constant, which describes equilibrium partitioning of a chemical between solution in water and the gas phase of the chemical, is the most relevant parameter to estimate the tendency of a chemical to volatilize from a surface impoundment or water.

For wastes which have been deposited in landfills, mixed in the ground, or have seeped downward from soil surface contamination, Henry's Law Constant, indicates the tendency of the chemical to partition between soil water in the vadose zone or ground water and the soil vapor phase. This partitioning is the first step for volatile air emissions from hazardous wastes beneath the soil surface. The vapor pressure and Henry's Law Constant, however, are not sufficient to provide a good indication of the magnitude of an air emission problem from volatile chemicals. Site-specific characteristics must be considered.

The environmental characterstics of the site are a major factor influencing the potential for, and extent of, an air emission problem. Soil characteristics such as porosity, moisture, and organic content are particularly significant when evaluating volatile emissions from land. Adsorption of a chemical to the soil reduces the extent of volatilization. Precipitation and downward movement also decrese the concentration of the chemical which will reach the air. Meteorologic conditions such as temperature, wind speed, and barometric pressure may influence emission rate from waste sites; other meteorologic characteristics such as wind speed and direction influence the movement of the chemical once it is released and, ultimately, the concentration at biological receptors.

Analytic techniques selected for inclusion in this manual were based upon the accuracy of description of the phenomena and availability of input data. The selected emission rate estimation methods for various types of hazardous waste sites include most of the methods selected by the Environmental Protection Agency. These approaches generally provide conservative estimates of downwind conditions that would not be expected to be exceeded.

Risk Determination

The appraisal of the adverse impacts of toxic substances on biological receptors at concentrations predicted to occur in the future is essentially identical to that employed to evaluate the adverse impacts of existing concentrations of toxic substances. Once a predicted level of contamination is determined, the AALs are employed to appraise the risks associated with the predicted levels of exposure. Should a significant risk be identified, a risk management process should be initiated.

COMPONENT V: Development of Mitigation Strategies and Remedial Action Selection

The Decision Tree Process comes to conclusion in this fifth component. Based on the degree of hazard and the characterstics of the site, alternatives for

remedial action can be identified. It is anticipated that the alternatives will be developed either by the responsible party or by regional contractors working for the State. When appropriate, State staff will develop a preferred alternative.

The objective of site mitigation is to assure that the biological receptors associated with each environmental pathway are not exposed to hazardous chemcials at levels above the Applied Action Levels (AALs). The strategies developed to achieve this objective may include control of the pathway (such as ground water extraction and treatment), modification of the pathway (such as capping a site to reduce infiltration), or control of the source material (such as on-site stabilization or treatment of contaminated soils). The physical, legal, and administrative actions necessary to implement site mitigation and maintain the desired effects of the site mitigation strategy are developed in the Remedial Action Plan (RAP). State staff and regional contractors will evaluate likely remedial alternatives.

Selection of the preferred remedial action should be made based on the scientific and technical evaluations cited in the preceding text. However, local, political, social, and other considerations must also be factored into the final decision. For instance, if a small site of marginal threat to health and environment exists within a widely contaminated industrial setting, the decision makers must consider if the public's best interests are being served through the implementation of an extensive remedial action. Factors that need to be addressed include the availability or unavailability of resources to mitigate other sources of contamination that are of comparable or greater significance, costs of materials and required manpower, the costs of transportation and disposal if soil or water removal is an option, and exposures likely to occur resulting from uncovering buried wastes.

The utilization of the Decision Tree Process in development of site mitigation strategies and RAPs will include a re-evaluation of the site to determine if post-mitigation exposure to hazardous chemicals associated with a particular site will exceed AALs.

CONCLUSION

The California Site Mitigation Decision Tree provides a systematic method for identifying and evaluating the risk associated with abandoned or uncontrolled hazardous waste sites. Because of the rapidly changing nature of scientific and technical knowledge in this area, the Decision Tree process has been designed for flexibility and expandability.

The Decision Tree process provides State decision makers with a logical, systematic, and time efficient approach to mitigating contaminated sites. This represents significant progress in meeting the challenge of protecting the public health and the environment from adverse effects of exposure to toxic chemicals found on these contaminated sites.

I. PRELIMINARY SITE APPRAISAL

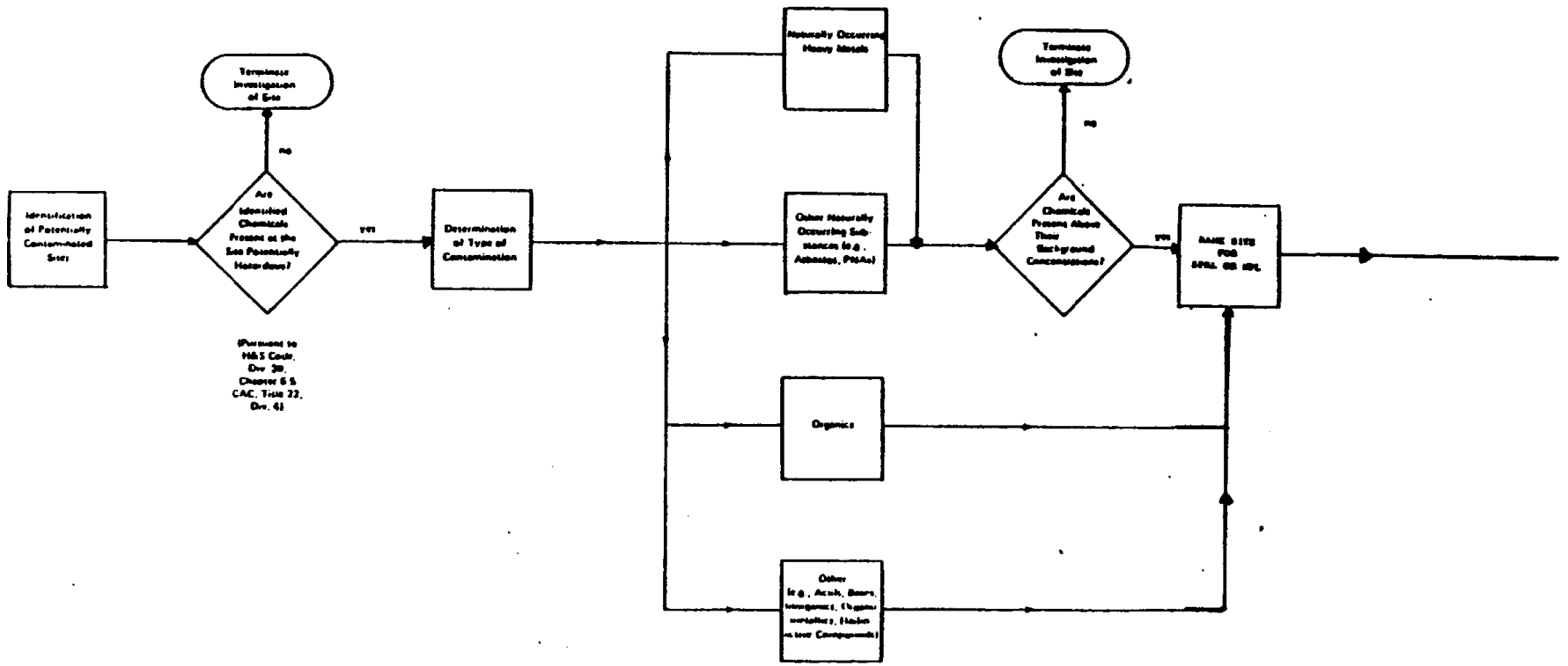
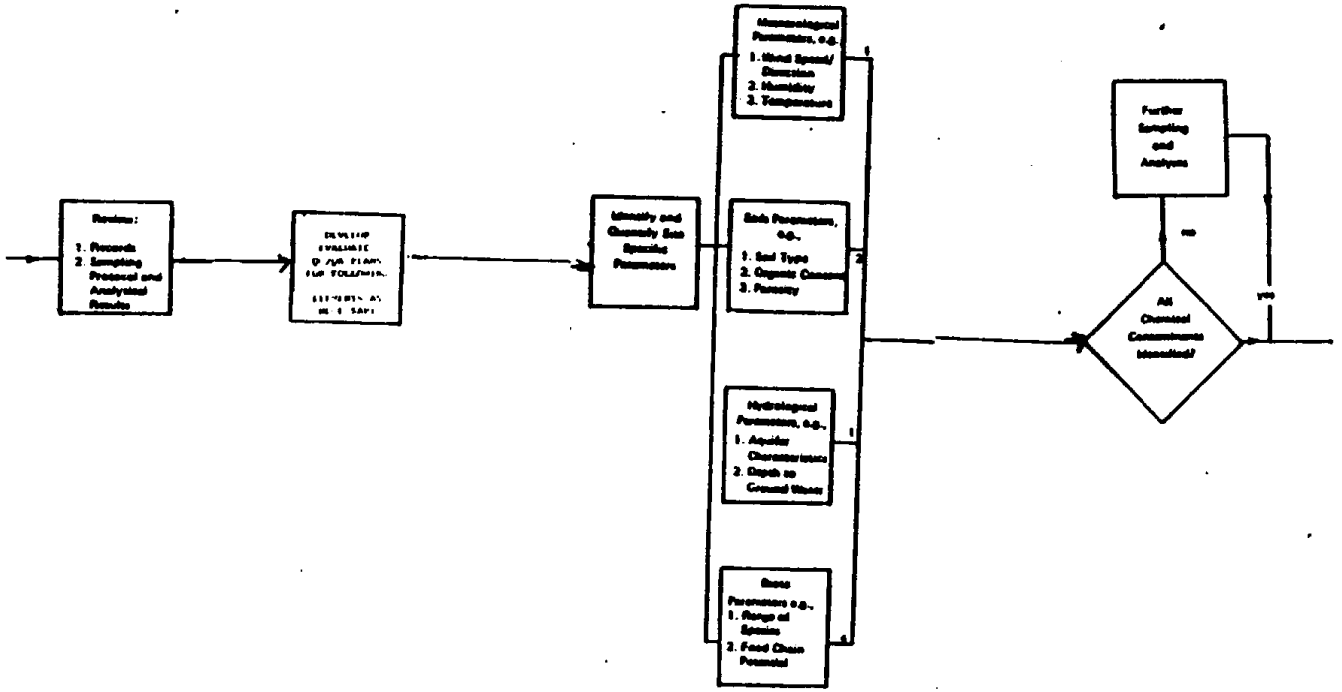
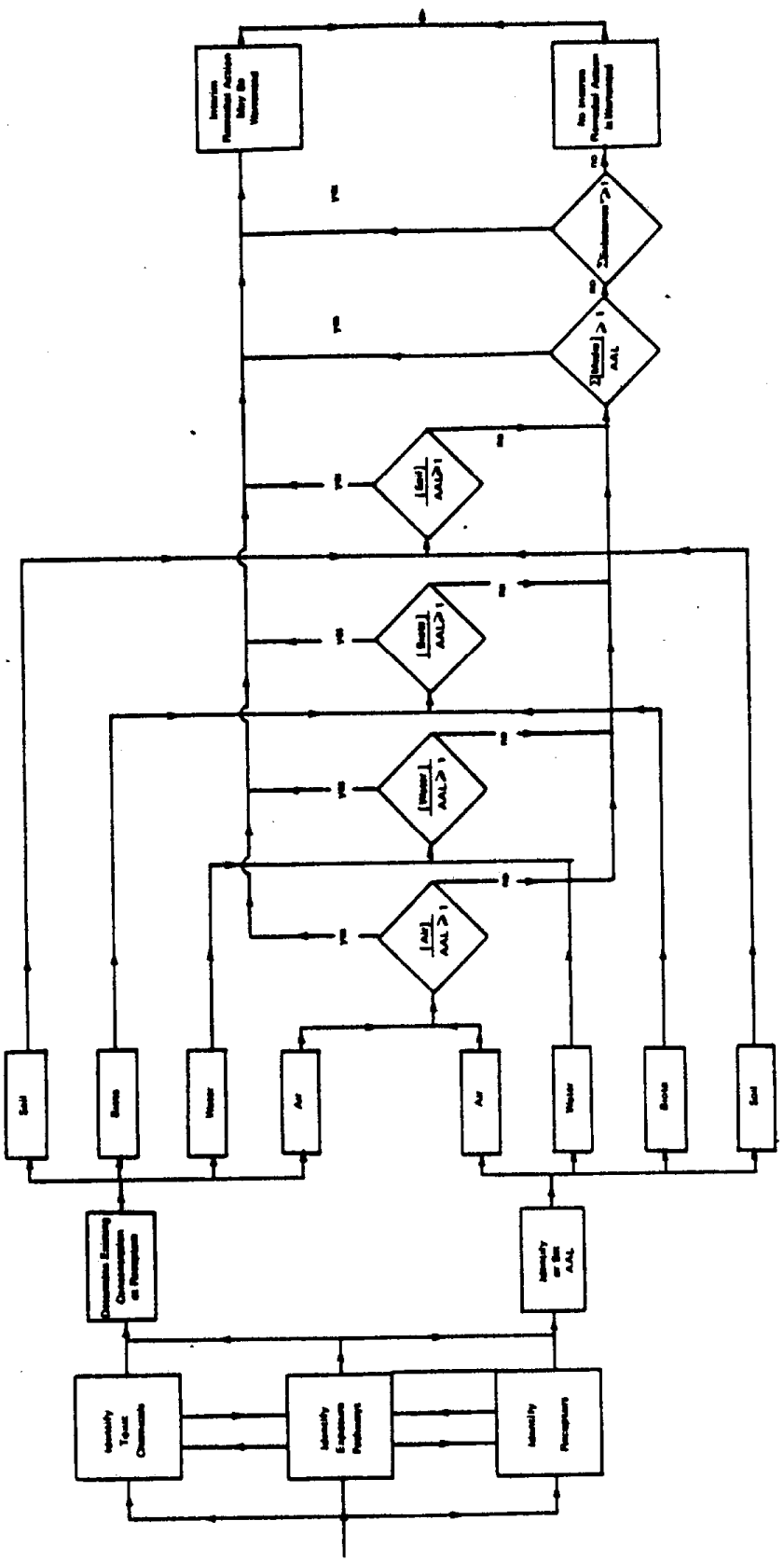
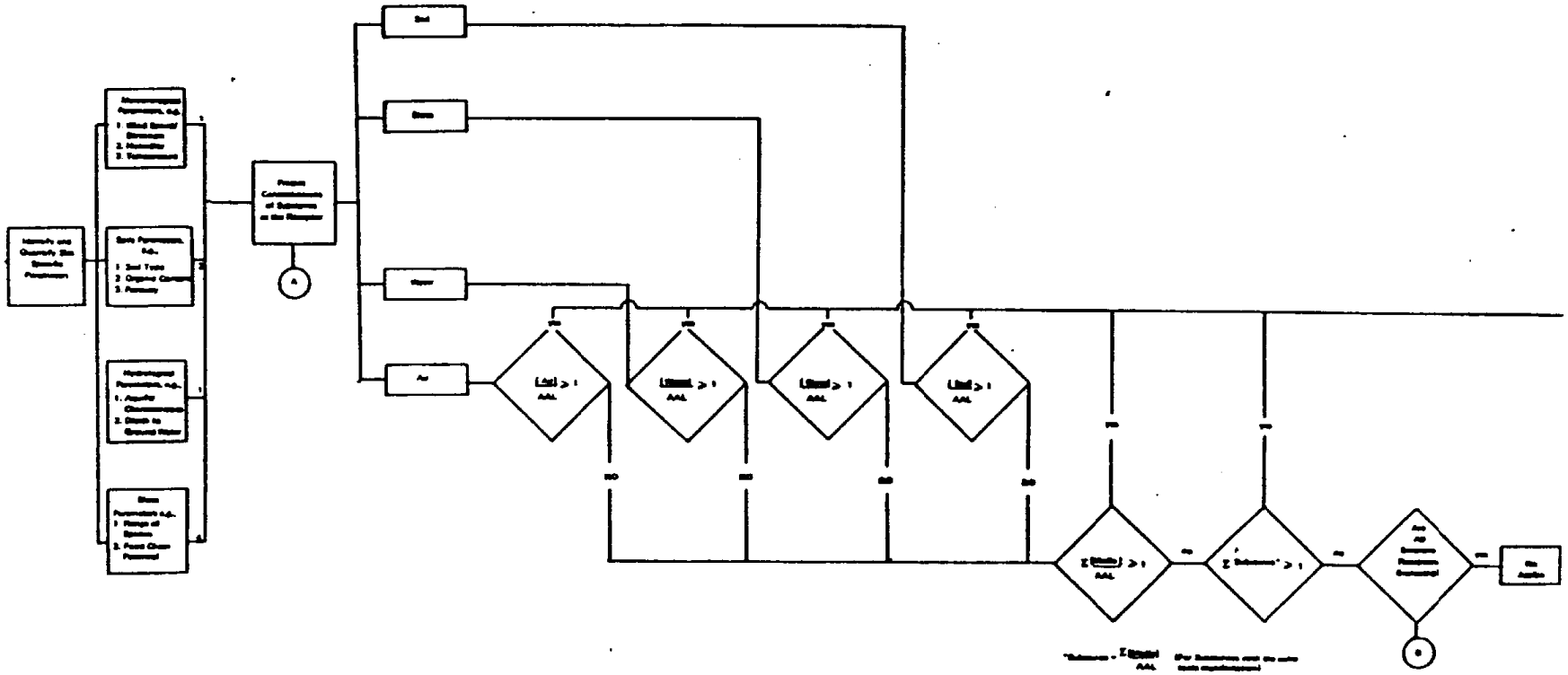


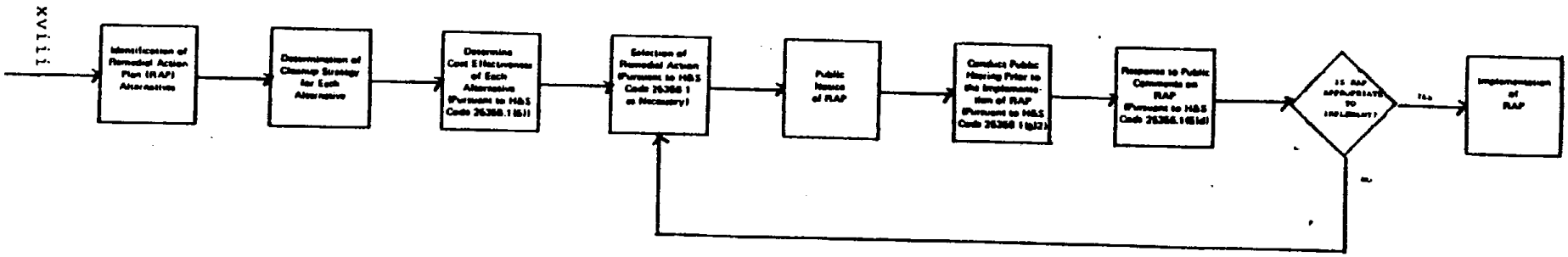
FIGURE 1. OVERVIEW OF THE DECISION TREE


Review:
1. Records
2. Sampling Protocol and Analytical Results
Identify and Quantify Site Specific Parameters
Meteorological Parameters, e.g.
1. Wind Speed/ Direction
2. Humidity
3. Temperature
Site Parameters, e.g.,
1. Soil Type
2. Organic Content
3. Porosity
Hydrological Parameters, e.g.,
1. Aquifer Characteristics
2. Depth to Ground Water
Biota Parameters e.g.,
1. Range of Species
2. Food Chain Potential
Further Sampling and Analysis
no
yes
All Chemical Contaminants Identified?





IV. ENVIRONMENTAL FATE AND RISK DETERMINATION



V. DEVELOPMENT OF MITIGATION STRATEGY AND SELECTION OF REMEDIAL ACTIO