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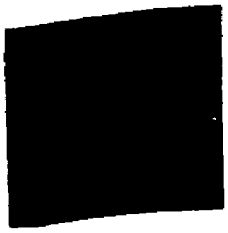

State-of-the-Art Review: PCDDs and PCDFs in Utility PCB Fluid

Prepared by
SCS Engineers, Inc.
Long Beach, California

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ABSTRACT

An extensive literature search was conducted into the chemical, environmental, toxicological, and analytical properties of chlorinated dioxins and dibenzofurans, particularly as these relate to PCB fluids. The literature search plus consultations with the leading international researchers in the field was used to prepare a timely state-of-the-art compilation of knowledge and theory on these important compounds. The chemistry of these compounds suggests that while chlorinated dibenzofurans can form directly from PCBs, chlorinated dioxins cannot. Both, however, can be formed from chlorophenols or chlorinated benzenes. Formation within electrical equipment PCBs under normal operating conditions is not likely. There has been little research conducted into degradation techniques. Some chemical degradation techniques and photolysis show promise. These compounds are extremely persistent in the environment, especially if protected from the sun. They are immobile in soil and sediment and do not biomagnify to any great extent. On a molecular basis, 2,3,7,8-TCDD is one of the most toxic synthetic molecules known to man, although its toxicological effects in man are still not well understood. Chemical analysis of dioxins and dibenzofurans is still a developing science. All analytical methods rely on some form of gas chromatography/mass spectroscopy. A further development which shows some promise as a rapid screening test is enzyme bioassay.

EPRI PERSPECTIVE

PROJECT DESCRIPTION

There has been inference and speculation about the potential presence and formation of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in polychlorinated biphenyls (PCBs). This conjecture arises from the identification of these materials in combustion by-products of fires associated with PCB equipment. There has not been a consolidation or review of the pertinent chemistry and toxicology literature from which one could separate legitimate scientific conclusions from unproven speculations and myths.

This final report for RP1263-11 endeavors to fill this need for a scientific literature review and serves as a literature basis on which subsequent experimental work may be developed. There are plans for a jointly funded project by two of EPRI's divisions--Electrical Systems Division and Energy Analysis and Environment Division--to investigate current analytic procedures, to analyze for specific PCDFs and PCDDs in electrical equipment fluids, and to attempt to experimentally define the conditions in electrical equipment that might lead to formation of PCDFs and PCDDs.

PROJECT OBJECTIVES

This project was conceived to examine and summarize the state of the art of knowledge regarding PCDDs, PCDFs, and other cyclic hydrocarbons in utility PCB fluids. The study also was to determine what conclusions could be made about formation conditions and to define gaps in experimental data.

PROJECT RESULTS

There are a number of important conclusions from this study. These are:

1. The association of PCB equipment and fires leads to formation of PCDFs and PCDDs.
2. While PCBs can lead to PCDFs, the formation of PCDDs from PCBs is unlikely.
3. PCDDs can form from chlorophenols.

4. PCDDs can form from chlorobenzenes in air at temperatures between 180 and 620°C.

5. PCDFs optimally form from PCBs at temperatures between 270 and 300°C and probably decomposes at temperatures above 300°C.

6. In unused PCBs, PCDF levels as high as 20 ppm have been noted.

7. After a fire, levels of PCDDs are less than 5% of the levels of PCDFs measured.

8. While sparking or soldering does not seem to cause PCDF formation from PCBs, welding creates an increase of a factor of 1.4 to 5 times the original levels.

9. There are no data regarding fires and contaminated mineral oil.

This report is of interest and value to utility and nonutility personnel who are faced with the issue of PCDFs and PCDDs in and around PCB equipment and who need to know about analyses and toxicology. It is also useful to those who must respond to questions, since the report summarizes what is known and identifies areas where additional research is needed.

A bibliography is provided for those who wish further details. This information should provide sufficient background for those concerned about the presence of dioxin and dibenzofuran in electrical equipment. The next step, which will be addressed by other divisions at EPRI, will measure levels of PCDFs in old equipment and will attempt to simulate extreme conditions under which they might form. The Coal Combustion Systems Division plans to document cleanup techniques that are used after a PCB fire.

Ralph Y. Komai, Project Manager
Coal Combustion Systems Division

ACKNOWLEDGMENTS

This report on chlorinated dibenzofurans and dibenzo-p-dioxins could not have been compiled without the aid and cooperation of numerous people. Chief among them was Christoffer Rappe, who provided data, figures, comments, and counsel. Others who have particularly aided in the task of identifying pertinent technical publications and data sources, as well as providing expert comments on this subject, include Bengt Ahling, Hans R. Buser, Gangadhar Choudhary, Paul Des Rosiers, Dennis Foent, Anders Gara, Lennart Hardell, Otto Hutzinger, Lawrence H. Keith, Ernest Merian, and Thomas O. Tiernan. Providing the utility and EPRI perspective we thank Gil Addis, Jacques Guertin, Harry Onishi, W. Corey Trench, John P. Woodyard, and Walter Weyzen for their review comments. Thanks also to the EPRI Project Manager, Ralph Komai, for constructive guidance in formulating and executing the project and presenting the information.

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EXECUTIVE SUMMARY

Since 1979, research has shown a potential concern resulting from the presence of chlorinated aromatic hydrocarbons associated with PCBs, predominantly polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and other related compounds. These compounds occur in extremely low concentrations in PCB liquids. However, some PCDD and PCDF isomers are orders of magnitude more toxic than PCBs.

There are a limited number of direct precursors and reactions by which PCDDs can be formed. In order to be a dioxin precursor, a compound must meet two conditions: (a) the compound must be an ortho-substituted benzene ring in which one of the substituents includes an oxygen atom directly attached to the ring; and (b) it must be possible for the two substituents, excluding the oxygen, to react with each other to form an independent compound.

The mechanisms by which PCDDs are formed during combustion are not well understood. Two suggested sources are (a) condensation of chlorophenols; and (b) thermal synthesis from organic materials and inorganic chloride (the "trace chemistries of fire" hypothesis).

There has been some question regarding the role of PCBs in PCDD and PCDF formation. Since PCBs do not fit the precursor model, PCDDs are not expected to form readily in a mixture of biphenyl compounds. The analysis of soots resulting from incidents involving PCDDs and PCDFs indicate the possibility of PCB conversion to PCDF or chlorobenzene conversion to PCDD and PCDF in a malfunctioning transformer or capacitor, provided that there is sufficient time at optimum temperature and/or oxygen conditions for their formation. Ordinary working conditions do not appear to be conducive to PCDD/PCDF formation.

Electrical equipment PCB fires may begin internally or externally. If they begin internally in an airless transformer or capacitor, the early stages are caused by electrical energy in internal arcing. Under these conditions, the major combustion

products are PCBPs; smaller amounts of PCDFs, PCPYs, and PCCYs are also found. If the equipment explodes or cracks and the PCBs are exposed to air, higher concentrations of PCDFs will form. If the fire begins externally, PCDFs are the major combustion product, accompanied by PCPYs and PCCYs; no PCBPs will form. PCDDs will form only if chlorobenzenes/chlorophenols are present in the PCB or askarel.

The following chemical degradation methods have been successfully tested on TCDD: oxidation by ozone, chlorine, or chloroiodides; alkaline dehydrochlorination; catalytic reduction with iron chlorides; treatment by ruthenium tetroxide; and micellar catalysis using chloroiodides.

PCDDs and PCDFs are also susceptible to photolysis. Investigations have also shown that dioxins exhibit a relatively strong resistance to biodegradation. It appears that the predominant mode of elimination of TCDD in soils is photodecomposition by sunlight, rather than microbial degradation. In aqueous ecosystems, 2,3,7,8-TCDD may be metabolized, but the rate is extremely slow.

Little is known of the environmental behavior of most of these compounds; only 2,3,7,8-TCDD has been studied in any detail. In general, TCDD is highly persistent in soils, where it is protected from the influence of solar photolysis and volatilization. It is virtually immobile in all but very sandy soils. The role of erosion as a transport mechanism is unknown. TCDD is not taken up by plants to any significant degree, and there is relatively little biomagnification in mammals. In water, TCDD is found to be strongly adsorbed onto bottom sediments.

On a molecular basis, 2,3,7,8-TCDD is one of the most toxic synthetic molecules known to man. Both higher and lower chlorination reduce toxicity, but the toxic symptoms of the various isomers are similar, the only difference being the amount required to produce a given effect.

The PCDD and PCDF isomers with the highest acute toxicity are those with chlorines in at least the 2,3,7,8 positions. Most of the research to date has been conducted on the toxicology of 2,3,7,8-isomers in animals. The toxic effects induced by 2,3,7,8-TCDD in all animal species included progressive weight loss and general weakening, with death occurring within a few weeks or months.

2,3,7,8-TCDD and other halogenated PCDDs exert their toxic effects by several mechanisms. They were primarily found to stimulate a number of enzyme activities (most

notably within the liver), primarily hepatic δ-aminolevulinic acid (ALA) synthetase and aryl hydrocarbon hydrolase (AHH). The PCDDs which have been found to induce ALA synthetase have two common properties: (1) halogen atoms occupy at least three of the four ring positions (i.e., 2, 3, 7, 8); and (2) at least one free nonhalogenated carbon atom is unoccupied. 2,3,7,8-TCDD has been found to be many orders of magnitude more potent than any other known inducer of ALA.

As a result of the various accidents in which humans were exposed to PCDDs and PCDFs, some epidemiological information is available on human toxicity. Chloracne, an often disfiguring and persistent dermatological disorder, is the most common indicator of poisoning in the human subject.

Chemical analysis of PCDDs and PCDFs is a complex task, dictated by (1) the large number of isomers of each compound (i.e., 75 PCDD isomers and 135 PCDF isomers), and (2) the difficulty of seperating and identifying isomers within a given chlorination class. In addition, many of these isomers have not been prepared in pure form; even the prepared ones have not been widely available.

Prerequisites for successful chemical analysis include representative sampling and good storage procedures; requirements for successful analysis include efficient extraction, sample purification (containment), good separation, ultrasensitive detection and quantification, and most desirable confirmation.

There are several methods for extraction of PCDDs and PCDFs from different sample matrices. All of the methods entail the initial addition of internal analytical standards, followed by destruction of the sample matrix (where practical) or extraction with an organic solvent, partitioning of the PCDDs/PCDFs into an organic phase and polar constituents into an aqueous phase and discarding of the latter, and liquid chromatographic cleanup (and, in some cases, HPLC cleanup) of the extracts. The cleanup steps remove matrix components which may interfere with subsequent detection and quantification of PCDDs/PCDFs, such as lipids and most of the chlorinated hydrocarbon residues.

It is generally accepted that the only technique which has both the required sensitivity and specificity for reliable detection and quantification of PCDDs/PCDFs is coupled gas chromatography/mass spectrometry (GC/MS). The first attempt to apply mass spectrometry for quantitative determination of TCDD was the high-resolution mass spectrometric technique. The limitations of this technique are the inability

to (a) distinguish TCDD ions from those originating from certain PCBs, and (b) separate TCDD isomers.

Methods using GC/low-resolution MS utilizes open-tubular packed gas chromatographic columns capable of separating TCDD from other PCDDs of different chlorine content, but have little capacity for separating discrete TCDD isomers. Their specificity in isomer determinations is derived from the cleanup methods used.

The combined GC (packed column)/high-resolution mass spectrometric techniques are similar to the high-resolution MS technique, but have better resolution and sensitivity due to the high-resolution scanning over a narrower mass range and at a more rapid scan rate. A further enhancement of the capabilities of the GC/high-resolution MS technique was capillary GC columns. The use of capillary GC columns provided the capability to separate discrete isomers of the PCDDs/PCDFs and improved the ability to obtain accurate quantitative data on the low concentrations of PCDDs/PCDFs in environmental samples.

Considerable success has been recently achieved in the separation of tetra- and hexa-CDD isomers by HPLC fractionation of discrete isomeric compounds. This technique appears to be particularly powerful when used in conjunction with GC/MS techniques, and does not necessitete the use of capillary GC columns.

Confirmation of PCDDs/PCDFs can be based on the proper molecular ions and fragments, as well as the intensities of the ion clusters due to the chlorine isotopes. If a sample contains a multitude of PCDD/PCDF isomers, the positive identification of PCDDs/PCDFs could be confirmed by cluster analyses.

PCDDs, PCDFs, and PCBPs produce a similar and characteristic pattern of toxic lesions, including hyperkeratosis and squamous metaplasia of the skin, and induction of a number of coordinetely expressed enzymes through stereospecific, reversible binding to a cytosolic receptor protein. These phenomena form the basis on which in vitro bioassey techniques have been developed for analyzing total PCDD/ PCDF biological activity, which in turn reflects their toxic potency. The significance of the bioassays, in addition to assessment of toxicological effects, is their usefulness as a rapid and reliable screen for detecting biologically active substances prior to chemical identification and quantification. The two most extensively studied bioassay techniques are aryl hydrocarbon hydroxylase (AHH) induction, and cell keratinization.

Future research is needed primarily into (1) the generation of PCDDs, PCDFs, and other related halogenated aromatics in utility PCB fluids, (2) chemical and bioassay methods for their analysis, (3) their toxicities, (4) their environmental fate, and (5) procedures for their destruction.

SUMMARY

Since 1979, research has shown a potential concern resulting from the presence of some chlorinated aromatic hydrocarbons associated with PCBs, predominantly polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and other related compounds, such as polychlorinated biphenylenes (PCBPs), polychlorinated pyrenes (PCPYs), and polychlorinated chrysenes (PCCYs). These compounds occur in extremely low concentrations in PCB liquids. However, some PCDD, PCDF, and PCBP isomers are orders of magnitude more toxic than PCBs.

There have been a number of incidents involving these compounds, including fires, industrial accidents, and contamination incidents resulting from inappropriate waste disposal. Several of these incidents have involved PCB-filled electrical transformers and capacitors. In each case, the electrical equipment was caught in an intense fire. The temperatures reached were extreme, far beyond the temperatures expected during normal (100° to 130°C), or even overload (200° to 250°C), conditions. Other incidents include a PCB-filled heat exchanger (in which the PCBs were again heated, probably in an oxygen atmosphere, for an extended period), an industrial accident involving the manufacturer of trichlorophenol, and an instance of chemical contamination from trichlorophenol manufacturing wastes.

CHEMISTRY OF PCDDs AND PCDFs

Very little published information is available on the chemical and physical properties of chlorinated dioxins, dibenzofurans, and related chlorinated cyclic hydrocarbons. They do not react with weak acids and bases and most redox agents, and are only slightly soluble in water and many organic solvents. In general, chemical stability increases with increasing halogen content.

There are a limited number of direct precursors and reactions by which PCDDs can be formed. In order to be a dioxin precursor, a compound must meet two conditions:

- The compound must be an ortho-substituted benzene ring in which one of the substituents includes an oxygen atom directly attached to the ring.

- It must be possible for the two substituents, excluding the oxygen, to react with each other to form an independent compound.

Although these conditions limit the number of direct dioxin precursors, there are many indirect precursors, i.e., compounds which do not fit the model, but can degrade or be transformed into direct precursors. The mechanisms by which PCDDs are formed during combustion are not well understood; thus, the effects of combustion temperature, fuel characteristics, catalysis, and other parameters have not been determined. Two suggested sources are:

- Condensation of chlorophenols.
- Thermal synthesis from any organic materials and inorganic chloride (the "trace chemistries of fire" hypothesis).

Pyrolysis (620°C) of chlorobenzenes in the presence of air yields PCDDs. Since chlorobenzenes do not fit the conditions set down earlier for dioxin precursors, it was postulated that the alkaline hydrolysis of chlorobenzene to PCDD involves a two-step condensation process consisting of dimerization of ortho-chlorinated phenoxy anions, followed by cyclization of polychlorinated 2-phenoxy phenate. This dimerization is exothermic. Below approximately 180°C, usually very minor amounts of PCDDs (<1 ppm) are formed, but significant quantities (1 to 10 percent yields) result at temperatures between 180°C and 620°C.

The high-temperature air oxidation of coal in the presence of chlorine, HCl, or NaCl has been shown to yield PCDDs. However, coal is a commercial source of benzene and cumene (a phenol raw material), so that formation of chlorinated benzenes and phenols is a probable intermediate step. Thermal decomposition of lignin and polyvinyl chloride (typical constituents of municipal refuse) can yield phenol, cresol, catechol, benzene, chlorobenzenes, HCl, etc. Thus, rather than just any organic material serving as a potential precursor, it seems more likely that those compounds which can be pyrolyzed to benzene, chlorobenzenes, phenol, catechol, and/or inorganic chlorine are more probable indirect precursors.

There has been some question regarding the role of PCBs in PCDD formation. In 1981, an electrical fire in the State Office Building in Binghamton, New York, resulted in the pyrolysis of PCB fluid from a transformer. Soot from this fire contained PCDFs and PCDDs. PCBs do not fit the precursor model discussed earlier, and PCDDs would not be expected to form readily in a mixture of biphenyl compounds. Conceivably, PCBs could be oxidized to polychlorinated hydroxybiphenyls, which could, in turn,

cyclize to form dioxins. Another reaction that has been suggested is the degradation of PCBs to chlorinated benzenes, subsequent oxidation to chlorophenols, and cyclization. However, neither mechanism quite fits the thermal decomposition pattern for PCBs.

The PCB fluid at Binghamton was a mixture of 65 percent Aroclor 1254 and 35 percent chlorobenzenes. It was suggested that pyrolysis of the chlorobenzenes is a more likely source of the PCDDs than PCBs. This reaction was discussed earlier.

While PCDFs and PCDDs can share some of the same precursors, PCBs are the most common source of PCDFs. PCDFs have been detected at levels up to approximately 20 ppm in a number of unused American, European, and Japanese commercial PCB mixtures. Concentrations and isomer ratios were found to vary with the type of PCB and the country of origin, probably as a result of differences in manufacturing conditions. Heating of PCB mixtures in air for 1 week resulted in the formation of PCDFs at temperatures over 270°C. Maximum levels were reached at 300°C, followed by declining levels at 330°C, suggesting decomposition. Primarily, di- and trichlorodibenzofurans were formed in this experiment.

TCDFs found in used transformer askarels ranged from approximately 0.004 to 4.7 ppm. The concentrations of TCDF in transformer askarels were found to increase with time in service. Furthermore, the apparent changes in TCDF concentrations caused by discharging or arcing were found to be negligible, most likely because of the short duration of the sparks or events causing failure. The amount of PCDDs, if detected, was usually less than 5 percent of the TCDF concentrations. Small amounts of terphenyl, quaterphenyl, and chlorinated terphenyl were also detected in some of the askarel samples, but they were not quantified.

Samples of used oil taken from two capacitors (Prodelec 3010) were analyzed for PCDFs; no apparent increase was detected. The formation of PCDFs when capacitor oils are exposed to sufficient oxygen and high temperatures was investigated by conducting laboratory tests with Prodelec 3010. The data indicated that neither sparking nor soldering resulted in an increase in the concentration of PCDFs; welding, however, resulted in a 1.4- to 5-fold increase in PCDFs.

The above findings indicate the possibility of PCB conversion to PCDF or chlorobenzene conversion to PCDD and PCDF in a malfunctioning transformer or capacitor, provided that there is sufficient time at optimum temperature and/or oxygen conditions

for their formation. Ordinary working conditions do not appear to be conducive to PCDD/PCDF formation. It should also be noted that most of the research on PCB/askarel conversion has been performed using pure PCBs/askarel conversion has been performed using pure PCBs or askarels containing at least 50 percent PCBs. The effects of dilution, as in contaminated mineral oil, have not been fully explored.

Fires in electrical equipment containing PCBs have also resulted in PCDF formation. Soot and wipe samples from PCB capacitor and transformer fires in the United States, Canada, Sweden, and Finland have all contained PCDFs.

Electrical equipment PCB fires may begin internally or externally. If they begin internally in an airless transformer or capacitor (with either no head space or an inert atmosphere), the early stages are caused by electrical energy in internal arcing. Under these conditions, the major combustion products are PCBPs; smaller amounts of PCDFs, PCPYs, and PCCYs are also found. PCBPs were identified for the first time in the aftermath of a 1981 capacitor explosion in Stockholm, Sweden.

If the equipment explodes or cracks and the PCBs are exposed to air, higher concentrations of PCDFs will form. If the fire begins externally, PCDFs are the major combustion product, accompanied by PCPYs and PCCYs; no PCBPs will form. PCDDs will form only if chlorobenzenes/chlorophenols are present in the PCB or askarel.

CHEMICAL AND BIOLOGICAL DEGRADATION

Very little research has been conducted on degradation mechanisms of dioxins and dibenzofurans. This is particularly true of the PCDFs; there is almost no information published on degradation. A number of researchers have speculated on degradation pathways, but little actual research has been conducted.

Several chemical degradation methods have been successfully tested on TCDD. Ozone bubbled through a suspension of TCDD in water and carbon tetrachloride for 50 hours achieved 97 percent degradation. Chlorolodides have been found to be effective in laboratory experiments, but tests on actual soil samples have not been highly successful. Alkaline dehydrochlorination has been tested on 2,4,5-trichlorophenol still bottoms containing up to 250 ppm TCDD. The reaction product was essentially free of 2,3,7,8-TCDD (ppt range) and other chlorinated dioxins (i.e., more than 99.95 percent destruction).

Experiments were also conducted on the separation of 2,4-D esters from 2,4,5-T in Agent Orange by fractionation, using catalytic reduction with iron chlorides. Overheads were found to contain 93 percent 2,4-D ester, whereas bottoms contained 93 percent 2,4,5-T ester. Ruthenium tetroxide was also found to destroy 2,7-DCDD and TCDD in laboratory tests. Micellar catalysis was tested using several chloroiodides. The most promising results have been obtained with benzalkonium dichloroiodide and cetylpyridinium dichloroiodide.

An experimental study was conducted to assess the feasibility of the chlorolysis process (i.e., chlorine gas as the oxidant at 600°C and 2500 psig) to convert typical chlorinated solvent wastes and Agent Orange to useful products such as carbonyl chloride, hydrogen chloride, and carbon tetrachloride. Agent Orange initially contained 14 to 18 ppm TCDD, whereas the carbon tetrachloride contained no measurable TCDD at a detection limit of 1 ppb.

PCDDs and PCDFs are also susceptible to photolysis. There are basically three conditions necessary for the photoreduction of chlorinated aromatic compounds:

- Presence of hydrogen donor, usually in the solvent.
- Ultraviolet (UV) light of appropriate wavelength.
- Absorption of the light.

In sunlight, only those compounds absorbing UV light above 290 nm are expected to be amenable to photodegradation.

There has been little research conducted on thermal destruction of PCDD, and even less on PCDF. Different researchers have reported 2,3,7,8-TCDD destruction at 800°C. In general, however, incinerator operating conditions currently considered adequate for TCDD destruction are a temperature of at least 1000°C and a dwell time of 2 seconds. At 700°C and a dwell time of 21 seconds, only 50 percent destruction was achieved; at 800°C and a dwell time of 21 seconds, 99.5 percent destruction was achieved.

Investigations have shown that dioxins exhibit a relatively strong resistance to biodegradation. Most of the work on dioxins has been focused on 2,3,7,8-TCDD because of its extreme toxicity. Approximately 100 strains of microbes, which had previously shown the ability to degrade persistent pesticides, have been tested. Only five strains were shown to have the ability to degrade 2,3,7,8-TCDD. It is possible

that the lesser chlorinated PCDDs are more susceptible to biodegradation, as is the case with PCBs. It also appears that the predominant mode of elimination of TCDD in soils is photodecomposition by sunlight, rather than microbial degradation. In aqueous ecosystems, 2,3,7,8-TCDD may be metabolized, but the rate is extremely slow.

ENVIRONMENTAL PERSISTENCE

With their high toxicities, the environmental fates of PCDDs and PCDFs are of increasing importance. However, little is known of the environmental behavior of most of these compounds. Only 2,3,7,8-TCDD has been studied in any detail, often with inconclusive results.

Primarily as a result of the 1976 PCDD incident in Seveso, Italy, most of the environmental research has centered on the fate of TCDD in soil. In general, TCDD is highly persistent in soils, where it is protected from the influence of solar photolysis and volatilization. It is virtually immobile in all but very sandy soils. The role of erosion as a transport mechanism is unknown. TCDD is not taken up by plants to any significant degree, and there is relatively little biomagnification in mammals. In water, TCDD is found to be strongly adsorbed onto bottom sediments.

TOXICOLOGY

On a molecular basis, 2,3,7,8-TCDD is perhaps the most toxic synthetic molecule known to man. Only bacterial exotoxins (i.e., Botulinum Toxin A, tetanus toxin, and diphtheria toxin) are more potent poisons.

Both higher and lower chlorination reduce toxicity, but the toxic symptoms of the various isomers are similar, the only difference being the amount required to produce a given effect.

The isomers with the highest acute toxicity are 2,3,7,8- tetra-CDD, 1,2,3,7,8-penta-CDD, 1,2,3,4,7,8-, 1,2,3,6,7,8-, and 1,2,3,7,8,9-hexa-CDD, 2,3,7,8-tetra-CDF, 1,2,3,7,8- and 2,3,4,7,8-penta-CDF, and 1,2,3,4,7,8- and 2,3,4,6,7,8-hexa-CDF. Most of the research to date has been conducted on the toxicology of 2,3,7,8-isomers in animals. The toxic effects induced by 2,3,7,8-TCDD in all animal species included progressive weight loss and general weakening, with death occurring within a few weeks or months. However, various experiments conducted to date have failed to provide the exact biochemical mechanisms of 2,3,7,8-TCDD lethality.

2,3,7,8-TCDD and other halogenated PCDDs were found to stimulate a number of enzyme activities, most notably within the liver. Hepatic δ-aminolevulinic acid (ALA) synthetase and aryl hydrocarbon hydrolase (AHH) were found to be induced by the presence of PCDDs, particularly 2,3,7,8-TCDD. The PCDDs which have been found to induce ALA synthetase appear to have two common properties: (1) halogen atoms occupy at least three of the four ring positions (i.e., 2, 3, 7, 8); and (2) at least one free nonhalogenated carbon atom is unoccupied. Available toxicological data indicate that those PCDDs that are not toxic generally do not induce ALA synthetase; those which are lethal, even at extremely low concentrations, induce the enzyme. 2,3,7,8-TCDD has been found to be many orders of magnitude more potent than any other known inducer of ALA.

Microsomal monooxygenase (e.g., AHH) is a membrane-bound enzyme complex which catalyzes the oxidative (and for certain substrates, reductive) metabolism of numerous xenobiotics and endogenous lipophilic compounds. 2,3,7,8-TCDD is a highly lipophilic macromolecule.

The induction of AHH and ALA by various PCDDs are carried out by the same mechanism. Theoretically, the most logical pathway of 2,3,7,8-TCDD metabolism is by hydroxylation at the 1, 4, 6, or 9 position of the molecule by AHH. The hydroxylation of aromatic rings in positions adjacent to halogen atoms is an uncommon reaction of the cytochrome P-450 monooxygenase system. This most likely accounts for the metabolic stability of 2,3,7,8-TCDD within mammalian systems. However, it has been postulated that 2,3,7,8-TCDD moves into a cell and binds to a specific cytosolic receptor. The receptor-dioxin complex then moves into the nucleus of a cell, where it initiates the synthesis of a specific messenger RNA, which directs the synthesis of cytochrome P-450. Other 2,3,7,8-TCDD molecules can then react with newly formed cytochromes, possibly to produce reactive intermediates that reduce or impair cellular metabolism, gradually inducing cell death. These metabolites may be excreted as innocuous by-products which may affect specific cells in other organs, or may act as carcinogens or cocarcinogens.

If the activity of AHH were increased, more 2,3,7,8-TCDD would be metabolized. If 2,3,7,8-TCDD, rather than a metabolite, is responsible for the toxicity, then increased metabolism by the P-450 monooxygenase system should lead to a decrease in toxicity. Conversely, if a metabolite(s) is responsible for the toxicity of 2,3,7,8-TCDD, then an increase in metabolism may result in an increase in toxicity. Several

studies have shown that 2,3,7,8-TCDD induces many enzyme systems while suppressing others. In addition to ALA and AHH, 2,3,7,8-TCDD induces the following enzymes:

- UDP glucuronyl transferase.
- Aldenyde dehydrogenase.
- Glutathione transferase B.
- DT-diaphorase.
- Benzopyrene hydrolase.
- Glutathione S-transferase.
- Ethoxycoumarin deethylase.

2,3,7,8-TCDD reduces the activity of the following enzymes:

- UDP-glucuronic acid pyrophosphatase.
- D-glucuronolactone dehydrogenase.
- L-gluconate dehydrogenase.

Another mechanism by which 2,3,7,8-TCDD may exert its toxic effect is by the increase of lipofuscin pigments. Lipid peroxidation, which precedes the formation of polymeric lipofuscins, is known to seriously damage membranous subcellular organelles, including lysosomes.

In one or more species of laboratory animals, bone marrow hypoplasia, testicular degeneration, renal pelvis and urinary bladder hyperplasia, and hemorrhage in the intestines and adrenals have been observed. Chronic administration of low levels of 2,3,7,8-TCDD to rats increases the incidence of neoplasia (tumors). The most malignant tumors were observed in the lungs, kidneys, liver, and skeletal musculature. Other neoplasms were found in the brain, skin, testes, and ears.

2,3,7,8-TCDD is a teratogen, interfering with only a few embryonic or fetal developmental processes. These teratogenic effects include impacts on hormones involved in reproduction, thymic atrophy, fatty infiltration of the liver, general edema, delayed head ossification, low birth weight, fetal reabsorption, and kidney abnormalities.

As a result of the various accidents in which humans were exposed to PCDDs and PCDFs, some epidemiological information is available on human toxicity. Chloracne,

an often disfiguring and persistent dermatological disorder, is the most common indicator of poisoning in the human subject. However, the absence of chloracne does not preclude intoxication.

Liver dysfunction is manifested by hepatomegaly and elevation of enzyme levels. Daily human exposure to 0.01 ug of 2,3,7,8-TCDD may result in incipient carcinogenicity, but existing evidence indicates that PCDDs are at most only mildly carcinogenic. Exposure to 4 ug of 2,3,7,8-TCDD would result in a shortened lifespan, and daily exposure to 290 ug would likely cause acute toxicity.

The immediate symptoms of exposure to dioxins include a burning sensation in the eyes, nose, and throat, followed by headaches, dizziness, nausea, and vomiting. Several days later, severe itching, redness, and swelling of the face (more marked over the eyelids, nose, and lips) may develop just prior to the onset of chloracne. It has been shown that as little as 20 ug of 2,3,7,8-TCDD on the skin can lead to chloracne. Within the initial weeks after exposure, inflamed nodules as well as pustules appear on the face, forearms, shoulders, neck, and trunk, leading to comedones and cysts. Sometimes chloracne is preceded by erythematous and edematous skin lesions. After 1 or 2 months, acneform eruptions emerge, and the skin becomes hyperpigmented. At approximately the same time, muscles, primarily in the thighs and chest area, become aggravated upon exertion. Insomnia, extreme fatigue, nervousness, irritability, and loss of libido also occur during this period.

Other symptoms of exposure include arthralgias, porphyria cutanea tarda, hyperlipidemia, cutaneous hyperpigmentation, and hirsutism. Porphyria cutanea tarda, a skin condition that usually occurs as a photosensitive dermatosis, is caused by the development of vesiculobullous lesions over exposed areas. The dermatosis is precipitated by minor trauma, and may result in areas of healed bullae, crusts, scars, and milia. Blurred vision and the loss of taste and smell may also occur.

There is no substantive evidence to indicate mutagenic, teratogenic, or embryotoxic effects on fetuses from 2,3,7,8-TCDD exposure in vivo. However, chromosomal abnormalities have been reported in in vitro cytogenic studies on human lymphocytes exposed to 2,4,5-T containing 0.09 ppm 2,3,7,8-TCDD. Chromatid breaks and deletions increased with increasing concentrations of 2,4,5-T. It was not possible to determine whether this was a toxic or genetic effect.

Analyses of liver, adipose tissue, and blood samples of both Japanese and Taiwanese patients suffering from rice oil disease ("Yusho") indicated that the highest concentrations of PCDFs were found in the liver. PCDF isomers, which were metabolized and excreted fairly rapidly (e.g., 2,3,6,7-tetra-, 2,3,4,6,7-penta-, 1,2,6,7,8-penta-, 1,2,3,4,8-penta-, and 1,2,3,4,6,7-hexa-CDFs), have two vicinal unchlorinated C-atoms in at least one of the two aromatic rings in the PCDF nucleus. In contrast, PCDF isomers retained in the bodies, such as 2,3,6,8-tetra-, 2,3,7,8-tetra-, 1,2,4,7,8-penta, 2,3,4,7,8-penta, 1,2,3,7,8-penta, 1,2,3,6,7,8-hexa-, and 1,2,3,4,7,8-hexa-CDFs, have lateral positions substituted with chlorine. The complete elimination of this compound from the organism is a function of its capacity to change macromolecules to more polar molecules.

In general, the clinical appearance of contaminated animals and the variety of lesions found during post-mortem examinations are identical for TCDFs, 2,3,7,8-TCDDs, and PCBs. The toxicity of TCDF is approximately 10 percent that of 2,3,7,8-TCDD, and 1000 times that of Arocior 1242. The correlation of metabolism with detoxification indicates that the greater toxicity of 2,3,7,8-TCDD versus TCDF could be the result of the relatively slower metabolism and excretion of 2,3,7,8-TCDD. Studies indicate that TCDF metabolism is carried out through a detoxification mechanism; species that are able to metabolize and purge TCDF are more resistant to its acute toxicity. The multiple-organ and histologic damage may be a result of both the relative rate of formation and the further inactivation of the reactive metabolites in different organs (e.g., liver, kidneys, stomach, etc.).

CHEMICAL ANALYSES

Chemical analysis of PCDDs and PCDFs is a complex task, dictated by (1) the large number of isomers of each compound (i.e., 75 PCDD isomers and 135 PCDF isomers), and (2) the difficulty of separating and identifying isomers within a given chlorination class. All classes are quite similar structurally, and thus similar in chemical and physical properties. In addition, many of these isomers have not been prepared in pure form; even the prepared ones have not been widely available. To date, the following isomers have been synthesized: the entire sets of tetra-, hexa-, hepta-, and octa-CDDs; 2 di-, 4 tri-, and 14 penta-CDDs; all tetra-CDFs; and some penta-CDFs.

Many of the early analytical studies focused on the determination of 2,3,7,8-TCDD because of its extreme toxicity. In addition, this PCDD isomer was one of the earliest available in sufficient quantity to use as an analytical standard. Some of this methodology was gradually adapted, modified, and expanded to accommodate

analyses of other PCDDs and PCDFs, although entirely new methods have also been developed.

Prerequisites for successful chemical analysis include representative sampling and good storage procedures; requirements for successful analysis include efficient extraction, sample purification (containment), good separation, ultrasensitive detection and quantification, and most desirable confirmation.

Sampling Methods

Sampling methods can markedly affect the results obtained. In general, the following dictates of good analytical practice should be applied in sampling: homogeneity of the sample matrix; collection of a truly representative sample; statistical considerations with respect to replicate analyses; and the resultant effects on precision and accuracy. In cases where samples are acquired by simple grab sampling, such considerations can usually be applied. In other instances (e.g., collection of flue gas samples from incineration sources), the sampling procedures are far less standardized, and their effectiveness and limitations have not been adequately evaluated.

Sample Extraction, Purification, and Separation

There are several methods for extraction of PCDDs and PCDFs from different sample matrices:

- U.S. Environmental Protection Agency (EPA): fish, adipose tissue, milk, water, soil, sediments.
- U.S. Food and Drug Administration (FDA): chemical formulations, fish.
- U.S. Fish and Wildlife Service: fish.
- Dow Chemical Company: fish, milk, fly ash, dust, urban particulates, municipal sludge, soil.
- Wright State University - Brehm Laboratory: soil, chemical waste, aqueous effluents, particulates.
- Umea University, Sweden: PCBs, fly ash, soot, wipe tests, blood.

All of the above methods entail the initial addition of internal analytical standards, followed by destruction of the sample matrix (where practical) or extraction with an organic solvent, partitioning of the PCDDs/PCDFs into an organic phase and polar constituents into an aqueous phase and discarding of the latter, and liquid

chromatographic cleanup (and, in some cases, HPLC cleanup) of the extracts. The cleanup steps are intended to remove matrix components, such as lipids, as well as most of the chlorinated hydrocarbon residues in the sample (e.g., PCBs, DDE, other common chlorocarbon residues, and fused-ring aromatic compounds), which may interfere with subsequent detection and quantification of PCDDs/PCDFs.

Instrumental Methods

Instrumental methods which have been utilized for detection and/or quantification of PCDDs/PCDFs in different sample matrices are as follows:

- UV Spectroscopy - TCDD.
- Thin Layer Chromatography - biological tissue; oils.
- Electron Capture/Gas Chromatography (EC/GC) - chemical formulations; PCDDs.
- Probe Mass Spectrometry (MS) - milk; animal tissue.
- Thermally Modulated EC Detector (TMECD) - animal tissue; chemical formulations.
- Packed Column GC/Low-Resolution MS (Selected Ion Monitoring) - chemical formulations; industrial hygiene samples.
- Packed Column GC/High-Resolution MS (Selected Ion Monitoring) - animal tissue; chemical wastes; aqueous solutions; soils; plant material.
- Capillary Column GC/High-Resolution MS (Selected Ion Monitoring) - human milk, animal tissue; water; sediments; combustion products.
- Capillary Column GC/Low-Resolution MS (Selected Ion Monitoring) - combustion products; oils; PCBs; human tissue.
- Liquid Chromatography Packed Column GC/Low-Resolution MS (Selected Ion Monitoring) - combustion products; isomer separation and identification.
- GC/Negative Chemical Ionization MS - animal tissue; wood; human tissue; combustion products.
- GC/Negative Ion MS - animal tissue; sediments.
- GC/Atmospheric Pressure Ionization MS - animal tissue; identification of isomers in standard mixtures.

Methods such as UV spectroscopy, thin layer chromatography, GC/EC detection, and TMECD have been developed for rapid screening of samples or qualitative detection.

It is generally accepted that the only technique which has both the required sensitivity and specificity for reliable detection and quantification of PCDDs/PCDFs is coupled gas chromatography/mass spectrometry (GC/MS). The first attempt to apply mass spectrometry for quantitative determination of TCDD was the high-resolution mass spectrometric technique. High-resolution scanning was accomplished over a narrow mass range (typically 0.3 AMU) at a rapid rate (4 scans/second). Due to the relatively high mass spectral resolution achieved, this technique was adequate for the purposes of distinguishing the molecular ion of TCDD from ions arising from the sample matrix (mostly biological samples). However, it was soon realized that the TCDD ions could not be distinguished from those originating from certain PCBs, if the latter were present in the sample extract in large concentrations relative to the TCDD (ppm versus ppt). Another limitation of the latter method was its inability to separate TCDD isomers.

GC/low-resolution MS utilizes open-tubular packed gas chromatographic columns. This method was capable of separating TCDD from other PCDDs of different chlorine content, but had little capacity for separating discrete TCDD isomers. The detection limits achieved in these determinations were in the range of 0.001 to 0.1 ppm. Their specificity in isomer determinations was derived from the cleanup methods used. Furthermore, these methods required that the analyte exhibit the appropriate GC retention time and a mass spectral response at the appropriate ion masses corresponding to TCDD.

The combined GC (packed column)/high-resolution mass spectrometric techniques were developed for determining TCDD in beef tissues and related environmental samples. These mass spectrometric methods were similar to the high-resolution MS technique, but were superior in that high-resolution scanning was generally accomplished over a narrower mass range and at a more rapid scan rate. This improved both the resolution capability and the sensitivity of the technique.

A further enhancement of the capabilities of the GC/high-resolution MS technique for analysis of PCDDs/PCDFs entailed the incorporation of capillary GC columns. The use of capillary GC columns provided the capability to separate discrete isomers of the PCDDs/PCDFs. The use of capillary column GC/high-resolution MS also improved the ability to obtain accurate quantitative data on the low concentrations of PCDDs/PCDFs in environmental samples.

Several laboratories have increased the use of capillary column GC/low-resolution MS in order to provide data on a wide range of PCDD/PCDF isomers in various types of samples, including PCB electrical fluids.

Considerable success has been recently achieved in the separation of tetra- and hexa-CDD isomers by HPLC fractionation of discrete isomeric compounds. This technique appears to be particularly powerful when used in conjunction with GC/MS techniques, and does not necessitate the use of capillary GC columns. No data on separation of PCDF isomers by this method have been reported.

Recently, several newer mass spectrometric techniques have also been investigated for the analysis of PCDDs/PCDFs. Among these are negative ion MS, negative ion chemical ionization MS, and atmospheric pressure/ionization MS. Results obtained thus far show that all of these offer enhanced sensitivity, selectivity, and specificity.

Quantification

To date, quantification has been based on peak area measurements by comparison with either internal standards or a calibration curve of known quantities of authentic standards (external standards). It has generally been assumed that all isomers of a particular PCDD or PCDF have the same response using the MS quantification. Two recent studies on the responses of 28 and 20 different PCDF isomers both showed a 3-fold variation in the response using an electron impact (EI) mode. Application of the negative chemical ionization (NCI) mode, however, resulted in 20-fold and 25-fold variations, respectively. For the higher chlorinated homologues, the differences appeared to be much smaller.

Confirmation

Confirmation of PCDDs/PCDFs can be based on the proper molecular ions and fragments, as well as the intensities of the ion clusters due to the chlorine isotopes. Moreover, if a sample contains a multitude of PCDD/PCDF isomers, the positive identification of PCDDs/PCDFs could be confirmed by cluster analyses.

For adequate confirmation of PCDDs/PCDFs, there are several parameters available. Compared to an authentic standard, the PCDDs/PCDFs in the sample should have the same following parameters: retention volume in the column chromatography in the cleanup steps; retention time in the gas chromatographic separation; molecular

weight; fragmentation and chlorine cluster in the MS detection; and difference in response factors comparing the EI and the NCI modes. These parameters, in addition to access to well-defined weighted standards, give the possibility of reliable analysis of PCDDs/PCDFs.

BIOASSAY ANALYSES

As discussed earlier, numerous clinical biomedical parameters have been shown to be altered in animals and humans receiving different doses of PCDDs, PCDFs, and PCBPs. In a number of investigations, 2,3,7,8-TCDD served as a prototype of individual PCDD and PCDF congeners and their mixtures. All of these compounds produce a similar and characteristic pattern of toxic lesions, including hyperkeratosis and squamous metaplasia of the skin, and induction of a number of coordinately expressed enzymes through stereospecific, reversible binding to a cytosolic receptor protein. These phenomena form the basis on which in vitro bioassay techniques have been developed for analyzing total PCDD/ PCDF biological activity, which in turn reflects their toxic potency.

The significance of the bioassays, in addition to assessment of toxicological effects, is their usefulness as a rapid and reliable screen for detecting biologically active substances prior to chemical identification and quantification. The discussion that follows focuses on two of these bioassay techniques: aryl hydrocarbon hydroxylase (AHH) induction, and cell keratinization.

Aryl Hydrocarbon Hydroxylase (AHH) Induction

The monooxygenase enzyme system, consisting of NADPH-cytochrome P-450 reductase and cytochrome P-450, is an integral part of the smooth endoplasmic reticulum, and catalyzes the catabolism of lipophilic compounds to more polar metabolites.

The induction of the hepatic monooxygenases by 2,3,7,8-TCDD occurs in most tissues of most species. These bioassays are extremely sensitive to a variety of chlorinated aromatic hydrocarbons. A large number of specific enzymes have been assayed; the most extensively studied of them is AHH.

AHH activity is a measure of cytochrome P-448. The induction of AHH is mediated by the reversible stereospecific binding of halogenated aromatic hydrocarbons to a cytosol protein, the cytosol induction receptor, which mediates the nuclear uptake of the toxicant. The receptor is determined by the Ah locus in mice. The Ah locus,

and hence the cytosol receptor, controls not only the induction of AHH activity but also a battery of coordinately expressed enzymes.

Two lines of evidence suggest that toxic lesions produced by TCDD, as well as biochemical responses, are mediated by the cytosolic receptor. First, the binding affinities of a large number of halogenated aromatic hydrocarbons for the receptor correspond very closely with the ranking of their toxic potencies. Second, two toxic responses produced by TCDD in mice, thymic involution and cleft palate formation in fetuses, have been shown to segregate with the Ah locus.

Compared with classical polycyclic aromatic hydrocarbon inducers of AHH, such as 3-methylcholanthrene, 2,3,7,8-TCDD produces a parallel dose-response curve; both compounds produce the same maximum response in AHH activity. However, 2,3,7,8-TCDD is 3×10^4 times more potent than 3-methylcholanthrene as an AHH inducer.

The AHH induction bioassay utilizes a rat hepatoma cell line which possesses low basal AHH activity and is highly inducible for this enzyme. The amount of AHH induction is determined by the fluorimetric assay of 3-hydroxybenzo[a]pyrene formation from benzo[a]pyrene (the substrate for the AHH enzyme assay), or by the fluorimetric determination of 7-ethoxyresorufin O-deethylation. The binding of a specific ligand or mixture of ligands is determined by measuring the dose-dependent displacement of [^{3}H]-2,3,7,8-TCDD previously bound to the protein.

Toxicity to Cells Cultured in Vitro

One of the most characteristic responses to TCDD is hyperkeratosis of the skin. When XB cells derived from a mouse teratoma are cultured at high density (to prevent spontaneous diffentiation) along with lethally irradiated 3T3 cells, the addition of TCDD produces a dose-related keratinization response, as detected by red staining with Rhodanile blue. This is a direct effect of TCDD on XB cells, and will occur in the absence of the feeder cells if the teratoma cells are cultured in 3T3-conditioned media. XB cells contain the cytosol receptor, and respond to TCDD with a dose-related induction of AHH activity.

Other halogenated aromatic hydrocarbon congeners, including PCDDs, PCDFs, PCBPs, and azobenzenes, were also found to induce keratinization although to a lesser extent than 2,3,7,8-TCDD. Keratinization could also be induced by some nonhalogenated aromatic hydrocarbons, such as benzanthracene and 5,6-benzoflavone. Since 2,3,7,8-TCDD is the most potent, but not the only inducer of this hyperkeratinization response,

this effect is referred to as "dioxin-like" activity. Unrelated toxins, however, failed to produce keratinization in the X8/3T3 culture system. Furthermore, a correlation has been demonstrated between the degree of toxicity of various TCDD isomers and congeners and the extent of hyperkeratinization response.

The X8/3T3 system is extremely sensitive, inducing keratinization with as little as 3.2 picograms of TCDD. In addition, this system is particularly relevent as a model, since it deals with an induced differentiation of the intact cell using an endpoint (hyperkeratinization) known to be relevant to human exposure (i.e., chloracne).

RESEARCH NEEDS

Future research is needed primarily into (1) the generation of PCDDs, PCDFs, and other related halogenated aromatics in utility PCB fluids, (2) chemical and bioassay methods for their analysis, (3) their toxicities, (4) their environmental fate, and (5) procedures for their destruction.

Generation of PCDDs, PCDFs, and Related Compounds

Since available information on both conditions and quantities of formation is insufficient and often contradictory, future studies should focus on better delineating the generation of PCDFs, PCDDs, and PCBPs. Standard and non-standard operating conditions should be compared with optimum formation conditions to determne if formation is indeed possible, and if the necessary precursors exist in PCB fluids. Such tests would provide specific insights into the chemistry, mechanisms, and optimum reaction conditions favoring the formation of these compounds from PCB fluids.

The data on structure-biological activity relationships for 2,3,7,8-TCDD in laboratory animals are extensive, while other PCDD homologues have been studied to a lesser degree. Toxicological data for PCDFs and PCBPs are very limited, as are data on the synergistic effects of two or more of the above by-products. For these compounds, specific data are lacking on acute lethality, chronic toxicity, carcinogenicity, teratogenic and reproductive effects, mutogenicity, and immunotoxicity. There are significant gaps in the relationship between the receptor theory and the induction of chloracne. Data on such effects as the extent of enzyme induction through specific receptor binding and cell keratinization should be acquired through bioassay techniques.

Chemical and Bioassay Analyses

Several areas require additional research in order to improve the capabilities for analysis of PCDDs/PCDFs. Such areas include (1) synthesis and isolation of additional pure PCDD/PCDF isomer standards for use in calibration and methods development; (2) synthesis of additional compounds which are chemically similar to PCDDs/PCDFs, and which may represent interferences in PCDD/PCDF analysis (e.g., diphenylethers, hydroxy- and methoxy-DPEs, and PCBs) for methodology evaluation; (3) development of higher resolution GC and/or LC columns for isolation of PCDD/PCDF isomers, (4) research on more specific detection, quantification, and confirmation methods, including possibly positive and negative ion chemical ionization MS, atmospheric pressure ionization MS, and MS/MS; (5) research into an isomer-specific analytical method to differentiate between toxic and nontoxic isomers; and (6) research into rapid chemical and/or biological screening methods; and (7) standardization of sampling methods, such as wipe tests, and establishment of criteria to identify the significance of results obtained via different sampling methods.

Environmental Persistence

The environmental pathways of these compounds need to be better defined. Neither the pathways nor the rates (other than general overall rates) for their physical migration and decay have been determined with any certainty. Further research is needed on photolysis to define the role and abundance of hydrogen donors, and the exact conditions under which measurable photolysis will occur in air, water, and soil. The role of uptake/ metabolism of these compounds by biota and their resulting fate in both water and soil needs further investigation.

Destruction/Removal

Since these compounds are both persistent in the environment and toxic to animals and humans, more research needs to be directed toward the development of chemical, physical, and/or biological methods for effectively treating wastes containing PCDFs, PCDDs, and PCBPs, as well as soil and water contaminated with these by-products.

Section 1

INTRODUCTION

BACKGROUND

Following promulgation of the Toxic Substances Control Act by the U.S. Congress in 1976, the U.S. EPA issued restrictions in May 1979 on the manufacture and commercial use of polychlorinated biphenyls (PCBs). Until the above regulations were issued, many transformers and capacitors contained PCBs. It has been estimated that since 1932, 135,000 transformers containing PCBs have been put into service in the United States (1).

The production of PCBs in the United States ceased in 1977. While some PCB equipment has been removed or replaced, many PCB-containing transformers and capacitors are still in use. In addition, due to past manufacturing and transformer servicing practices, trace amounts of PCBs have been introduced into mineral oil-filled equipment. Hence, it is likely that, in addition to askarel transformers and capacitors, many of the oil-filled transformers currently in service contain PCBs in concentrations ranging in the low parts per million.

Since 1979, research has shown a potential concern resulting from the presence of some chlorinated aromatic hydrocarbons associated with PCBs, predominantly polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). Traces of similar compounds, such as polychlorinated biphenylenes (PCBPs), polychlorinated pyrenes (PCPYs), and polychlorinated chrysenes (PCCYs), have also been reported in some samples. These compounds occur in extremely low concentrations in PCB liquids. However, some PCDD, PCDF, and PCBP isomers are acutely toxic as determined by laboratory tests on animals and demonstrated in industrial incidents. Toxicities of some of these isomers are orders of magnitude higher than PCBs.

PCDD and PCDF compounds have been found in the original PCB fluids manufactured in the United States, Europe, and Japan. Several PCB accidents in which PCDDs and PCDFs were prominent have also been documented, including the Yusho incident in Japan involving ingestion of PCDF-contaminated rice oil, and a similar subsequent incident in Taiwan. More recently, a fire involving electrical equipment containing

PCB fluids in a Binghamton, New York, office building produced soot which was found to contain PCDFs and PCDDs. These findings indicate the possibility that PCDDs, PCDFs, and other related chlorinated aromatic compounds can form from askarel fluids at high temperatures.

PROJECT OBJECTIVES

The purpose of this study is to determine the state of knowledge on the formation of PCDDs, PCDFs, and other related chlorinated aromatics in on-line electrical equipment containing PCBs, and to provide EPRI with a technical planning tool for formulating its future research on this subject. The specific objectives of this study are as follows:

- The state of knowledge in the area of PCDDs/PCDFs in utility dielectric fluids.
- Recommend future research needs in the areas of PCDD/PCDF formation in utility electrical equipment, their chemical and biological analyses, environmental fate, toxicology, and techniques for their destruction.

Specific information in this report was collected by (1) literature search; (2) attendance at the 184th National Meeting of the American Chemical Society in Kansas City, Missouri (September 1982), and the 3rd International Symposium on Chlorinated Dioxins and Related Compounds in Salzburg, Austria (October 1982); (3) discussions with leading researchers (worldwide) in the subject area; (4) contacts with transformer service companies and manufacturers; and (5) discussions with representatives of the utility industry.

REPORT ORGANIZATION

Section 2 of this report details a number of incidents involving the generation of PCDDs and PCDFs. The physical and chemical characteristics of PCDDs and PCDFs, along with their formation chemistry and the chemistry of transformers/capacitors, are presented in Section 3. Physical, chemical, and biological degradation of these chemicals and their persistence in the environment are discussed in Sections 4 and 5, respectively. Toxicological effects on laboratory animals and humans are discussed in Section 6. The current status of analytical procedures for the determination of PCDDs and PCDFs is reviewed in Section 7. This review includes both chemical and bioassay analyses. In Section 8, recommendations are made for future research needs. These recommendations reflect the identified data gaps in each of the subject areas covered in Sections 3 through 7. Additional information pertinent to the topics discussed in this report is presented in Appendices A through D.

Section 2

ACCIDENTS INVOLVING GENERATION OF PCDDs/PCDFs AND OTHER RELATED CHLORINATED AROMATIC HYDROCARBONS

INTRODUCTION

In this section, we examine a number of incidents including fires, industrial accidents, and contamination incidents resulting from inappropriate waste disposal. The common thread running through these incidents is the production or release of polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs), some of which are extremely toxic (Figure 2-1). In some of the incidents, there is also evidence of the production of other chlorinated polynuclear aromatics, such as the polychlorinated biphenylenes (PCBPs) and polychlorinated pyrenes (PCPYs) (Figure 2-1).

A number of these incidents involve PCB-filled electrical equipment, including transformers and capacitors. In each case, the electrical equipment was caught in an intense fire. The temperatures reached were extreme, far beyond the temperatures expected during normal (100° to 130°C), or even overload (200° to 250°C), conditions. Other incidents include a PCB-filled heat exchanger (in which the PCBs were again heated, probably in an oxygen atmosphere, for an extended period), an industrial accident involving the manufacturer of trichlorophenol, and an instance of chemical contamination from trichlorophenol manufacturing wastes.

ELECTRICAL POWER EQUIPMENT EPISODES

Toronto, Canada

In December 1977, a transformer substation burned in Toronto, Canada. Very little has been published concerning this incident, but it has been cited that the PCB fluid originally contained 0.05 ppm PCDFs; the ash produced contained 5 ppm (2). No information was reported regarding distribution, health effects, or chemical analyses.

Norrtälje, Sweden

In September 1978, an intense fire burned a capacitor battery north of Norrtalje, Sweden. Eighteen PCB capacitors were burned to various degrees, and some exploded.

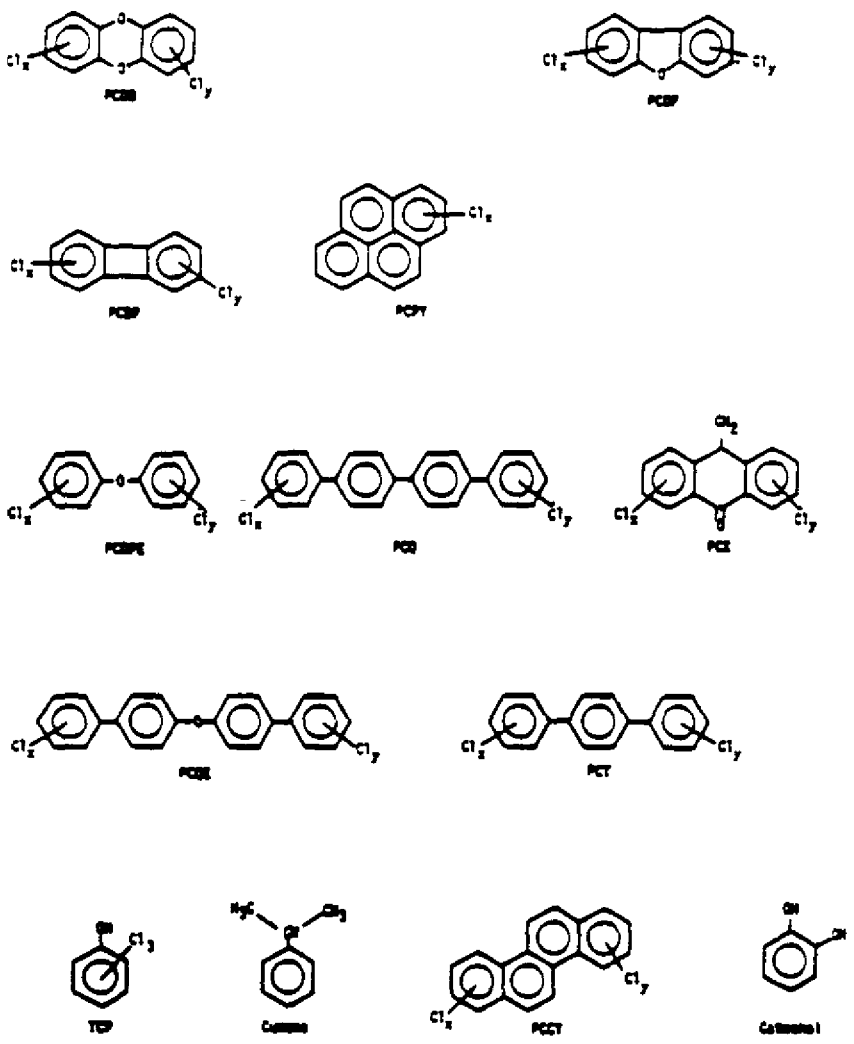


Figure 2-1. Pertinent Chemical Structures

Examination of the fluid again showed a large increase in PCDF concentrations (2, 3). Concentrations of PCDFs on nearby objects (fences and pine trees) were below detection limits. Samples of blood taken from firefighters showed PCB concentrations (2.3 to 3.6 ug PCB/g) within normal limits and no detectable PCDF (2).

Binghamton, New York

In February 1981, an intense fire occurred in the utility room of the 18-story Binghamton State Office Building (Binghamton, New York), resulting in the loss and apparent pyrolysis of 180 gallons of PCB fluid from a transformer (4, 5, 6). The fire was apparently electrical in origin, and produced a fine soot which was distributed throughout the building by the ventilation system (4).

Analysis of the soot revealed PCBs, PCDFs, PCDDs, and PCBPs. When PCDDs and PCDFs were discovered, cleaning was suspended indefinitely, and the building was sealed. Although the building was unoccupied at the time of the fire, over 450 persons claimed that they had been exposed to the chemicals, and many have filed suits against the state (6). No published description of the symptoms experienced by allegedly exposed persons was given.

Stockholm, Sweden

In August 1981, another electrical fire occurred in a 10-kV capacitor battery in a power station in Stockholm, Sweden. Like the Binghamton fire, this incident is thought to have been caused by a malfunction of the electrical equipment. Analysis of residues at Stockholm also showed PCDFs and strong evidence of PCBPs. Unlike the Binghamton fire, no PCDDs were found (3). No injury or illness was reported in connection with this fire.

Skovde, Sweden

In March 1982, a 400-V capacitor battery serving a high-frequency oven in a metal treatment factory burned in Skövde, Sweden (3). The capacitors involved were both mineral oil-containing and PCB types, and the fire appears to have been unrelated to equipment malfunction. Of 21 PCB-filled capacitors, 12 were ruptured during the course of the 2-hour fire, which reached at least 1080°C in some parts of the capacitor room. The smoke from the fire was distributed throughout the 100 x 200-ft (30 x 60-m) building.

Analysis of soot samples showed PCDFs, but no PCDDs or PCBPs. Another series of chlorinated compounds was detected; molecular weights and numbers of chlorines corresponded to polychlorinated pyrenes (PCPYs), but no confirmation or quantitation was possible due to the lack of standards (3).

NONELECTRICAL INDUSTRIAL EPISODES

Chick Edema

A. L. Young has reviewed the dioxin poisoning episodes known collectively as "chick edema" (7). During 1957, millions of chickens died after consuming feeds contaminated with 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin (HCDD). Clinical signs of the disease included labored breathing, unsteady gait, subcutaneous edema, and sudden death. The birds were found to have excessive fluid in the abdominal cavity and the pericardial sac, and swollen, palid livers. Mortality was reported to be as high as 50 percent. The contaminant, known as "chick edema factor," was isolated in the early 1960's, but the structure was not elucidated until 1967.

Chick edema disease was essentially dormant through the 1960's until another outbreak in 1969. On this occasion, it was learned that dioxins had accumulated in the edible tissues of the chickens. In each case, feeds had been supplemented with fats and fatty acids, generally by-products of stearic and oleic acid manufacture. It was speculated that the fats were contaminated with commercial chlorophenols, which were contamnated by or converted to dioxins during heating.

Yusho (Japan)

In October 1968, Japanese health officials became aware of an epidemic disease that came to be known as "Yusho." Victims exhibited such symptoms as acne-like skin eruptions, pigmentation of the skin and mucous membranes, swelling of the eyelids, increased eye discharge, hyperkeratotic plaque on the soles of the feet and palms of the hands, and hyperemia of the conjunctiva (8, 9).

The cause of the disease was eventually traced to certain lots of rice oil (Kanemi) produced during February and March 1968 (9). It is speculated that the oil was contaminated during vacuum distillation via pinhole leaks in the piping of a heat exchanger. The heat exchanger used PCB fluid as the heat exchange medium, and it maintained the distillation tank at 230°C. GC analyses showed 2000 ppm "Kanechlor" brand PCB fluid in the oil (9).

A 1971 report set the number of Yusho cases at 1001 (9). A subsequent report set the number at 1200 (10). Patients showed detectable concentrations of PCBs in their subcutaneous fat. Birth defects and retardation of normal growth were also noted. Of 13 babies born to women affected with Yusho, 2 were stillborn, 10 had hyperpigmentation, and 9 had increased eye discharge (8). Pigmentation disappeared in 2 to 5 months (11). Children initially exhibiting retarded growth caught up with a normal control group within a few years (12).

After the general potential for PCDF and PCDD contamination of PCBs was reported, used PCB fluid and the rice oil involved in the Yusho poisoning were reanalyzed. No significant levels of PCDD or PCBP were found (10). PCDF was found in both fluids (15.6 and 5.65 ug/g, respectively) (3, 10, 13, 14, 15).

Other analyses of the rice oil indicated that, in addition to the PCDFs present, there were also other chlorinated compounds, including quaterphenyls (PCQs), quaterphenyl ethers (PCQEs), and terphenyls (PCTs) (Figure 2-1). Most of the "unknown organo-chlorine compounds" were identified as polychlorinated quaterphenyls (PCQs), and most of these were hepta- or octachloroquaterphenyls (16).

Yusho (Taiwan)

Health authorities in Taiwan reported a second outbreak of Yusho in the Taichung and Changhua areas of Taiwan in 1979 (17, 18). It is reported that 1900 people were affected by PCB-contaminated rice oil, but no further information was given concerning the origin or production of the oil (17). The researchers presume that the oil was contaminated by the heat exchanger during manufacture (18). Analysis of the contaminated oil showed both PCBs and PCDFs (18). Blood samples from affected patients also showed PCBs (17) and PCDFs (19).

St. Louis, Missouri

Between February and October 1971, trichlorophenol (TCP) distillate residues from the Northeastern Pharmaceutical and Chemical Company (NEPACCO) in southern Missouri were collected by a salvage oil company in St. Louis (Figure 2-1). The 18,000 gallons (68,130 liters) collected were dumped into a large holding tank, along with other waste oil and sludge. During May and June of that year, the salvage company sprayed three horse arenas with waste oil from the tank to control dust. In subsequent weeks, birds, mice, cats, dogs, and horses exposed to these areas became sick and died. Among surviving horses that were pregnant, 26 abortions occurred, and many foals died soon after birth, some with manifest deformities (20, 21). Several

children exposed to the contaminated arenas became ill, with symptoms including chloracne, headaches, aching joints, and inflammation of the kidneys (21).

One little girl developed hemorrhagic cystitis following repeated exposure to one of the arenas. In 1977, Beale, et al., reexamined her and found that she had grown normally. Detailed physical, chemical, and neurological examinations of the child and her mother and sister (who were also exposed, but to a lesser extent) were normal (7)

Laboratory analyses of soil from one of these arenas showed approximately 5000 ppm TCP, 30 ppm TCDD, and 1350 to 1590 ppm PCB (21). 2,3,7,8-TCDD was found to be a major component of the PCDDs, and 1,3,7,8-TCDD a minor one; no tri- or penta-CDDs were detected (C. Rappe, Personal Communication). In addition to PCDDs, approximately 3 ppm PCDFs were found (mainly 2,3,6,8-TCDF, with some 2,3,7,8-TCDF and traces of penta- and tri-CDFs) (C. Rappe, Personal Communication). Furthermore, polychloroxanthenes (PCXs) (Figure 2-1) were found to be a major component (primarily 1,2,4,6,8,9-hexachloroxanthene, with traces of pentachloroxanthene) (C. Rappe, Personal Communication). It should be noted that hexachloroxanthene is a major impurity in commercial hexachlorophenes (C. Rappe, Personal Communication). The pharmaceutical company waste storage tank, left undisturbed, was analyzed in 1974, and showed approximately 300 ppm TCDDs, 30 percent TCP, and no PCBs (21). The PCBs in the soil apparently came from the wastes of another, unidentified company.

Following an anonymous phone call in 1979, the EPA began further investigations of NEPACCO's waste disposal practices. Eventually, 30 sites in the Spring River Basin of southwest Missouri were identified. Results indicated widespread distribution of 2,3,7,8-TCDD attributable to NEPACCO's waste disposal (22).

Seveso, Italy

One of the largest accidental TCDD incidents occurred in a TCP manufacturing plant north of Seveso, Italy, in 1976. The reaction went out of control, and the vessel vented to the atmosphere, releasing a caustic cloud which settled on several communities to the south of the plant. Because the exact nature of the chemical cloud was not known, the company tried to deemphasize the levels of toxic chemicals involved, and no immediate public health precautions were taken (7, 23, 24).

Within a week of the accident, widespread phytotoxicity (poisonous to plants) was evident, small animals were dying, and children were being admitted to hospitals

with burns. Tests confirmed that the chemical cloud consisted primarily of 2,4,5-trichlorophenol, contaminated with 2,3,7,8-TCDD and traces of 1,3,7,8-TCDD, 2,7- or 2,8-di-CDD, 2,3,7-tri-CDD, and mono-CDD (25). Minor amounts of tri- and tetra-CDF were also found in the first samples (25). The last sample was analyzed in March 1977 for the presence of other neutral and phenolic compounds. Apart from the PCDDs mentioned above, traces of di- and trichlorophenols, tetra- and hexachlorobenzene, PCB, and DDE were detected.

Areas where TCDD concentrations measured 0.001 ppm or more were evacuated (7). Children in other local areas were evacuated during the day; such areas were closed to nonresidents, and vegetable growing and animal breeding were forbidden (7, 23). Analyses indicated that 98 percent of the TCDD was sorbed in the top 4 cm of the soil (7). Estimates of the total TCDD deposited varied from 450 to 3000 g (7, 23).

Decontamination measures that were eventually taken included vacuuming, scraping, and washing of walls, floors, and ceilings; removal of most of the furniture; and removal of the top layer of soil in gardens and orchards.

Reports on health impacts resulting from the accident vary concerning the number of people affected. The number of chloracne cases ranged between 134 and 187, with most of the cases occurring in children (7, 26). Chloracne symptoms were rarely severe and seldom recurred. Where they recurred, it was usually in mild to very mild cases (7). Neurological damage was reported in 33 adults, but no liver or gastrointestinal damage was documented (27). Another report indicated no abnormal neurological findings, and no laboratory evidence of significant hepatotoxicity or deranged porphyrin metabolism (7). Analyses through 1980 indicated no significant changes in either the general or cause-specific mortality rate in the area (28).

FORMATION OF PCDD, PCDF, AND OTHER CHLORINATED ORGANICS

PCDD, PCDF, and other chlorinated organics have been formed in the aftermath of these accidents, and have been detected both alone and in various combinations. Table 2-1 summarizes the toxic products formed in each case. It is difficult to compare the results from each incident because of the different sampling methods and sample matrices (wipe tests, soot samples, etc.).

PCDD Incidents

PCDDs have been accidentally produced in the course of a few of these incidents, and under widely varying circumstances. The PCDDs (or simply "dioxins") found in the

wake of the explosion in Seveso have become a byword for industrial environmental contamination. As much as 5.5 to 6.5 lb (2.5 to 3 kg) TCDD were released (7), although some estimates are much lower. As previously stated, 98 percent of the TCDD is strongly sorbed in the top 1.5 in (4 cm) of soil. Ongoing decontamination efforts have been focused on removing topsoil, depositing it in the evacuated zone, and finding a way to decontaminate it, possibly through extended biological degradation (7).

Table 2-1

SUMMARY OF CHLORINATED AROMATIC HYDROCARBONS DETECTED DURING DIFFERENT ACCIDENTS

Accident	PCDDs	PCDFs	PCOs	PCOEs	PCTs	PCBPs	PCPYs	PCCYs	PCNs	PCDPEs	Reference
Toronto		5 ppm									2
Norrtalje (1978)		45-107 ug/g									2
Binghamton	2.8-19.9 ppm	124-2,163 ppm				•	•	•			4, 29
Stockholm (1981)		43-104 ug/g				•	•	•			2
Skovde		0.01-0.773 ug/m^2					•	•			30
Chick Edema	•										7
Yusho, Japan		1-18 ppm	•	•	•						14
Yusho, Taiwan		0.18-1.66 ppm	•		•						18
St. Louis	30 ppm	•							•		21
Seveso	0->1000 ug/m^2	•								•	31

• Quantification and confirmation not possible due to lack of standards

Seveso is the only TCDD incident for which "operating conditions" are known. Thermographic records at the plant showed an uncontrolled increase in the temperature of the reaction vessel, following failure of a safety shutdown device. The mixture was heated at 230 to 240°C for 4 to 5 hours and 3 to 4 atm before a safety disc ruptured, releasing nearly the entire mixture (7).

The waste oil, which contained still bottom residues from TCP manufacture and was used to control dust in Missouri horse arenas, contained sufficient TCDD to yield 30 ppm TCDD in a soil sample (21). When the waste oil storage tank was examined later, it was found to contain 300 ppm TCDD (21). As noted above, 2,3,7,8-TCDD has been found in disposal sites throughout the Spring River Basin. As of mid-1982, 30 possible sites had been identified, and investigations had accounted for approximately 110 lb of still bottom-derived TCDD.

In the Binghamton State Office Building fire, PCDDs were formed during the combustion of transformer fluid consisting of PCBs and chlorinated benzenes. The New York State Department of Health analyses found 2.8 and 2.9 ppm (ug/g) 2,3,7,8-TCDD in the soot when duplicate analyses were run on a single sample (4). Another sample of soot was analyzed and reported to contain 20 ppm PCDD and 0.6 ppm 2,3,7,8-TCDD (29). PCDDs were not found in the residues of the other transformer fires. Rappe, et al., attributes this difference to the presence of chlorinated benzenes in the dielectric fluid, as PCDD is known to be formed in the pyrolysis of chlorinated benzenes (3).

PCDF Incidents

PCDFs have been detected in more incidents than PCDDs; they have been found in all PCB fires in which measurements were taken, as well as in the Yusho poisoning incident. In the Binghamton transformer fire, 273 and 124 ppm PCDF were reported for duplicate analyses of a soot sample (4). It is thought that this represents 2,3,7,8-TCDF, but positive confirmation of the structure was not possible due to a lack of internal standards. Rappe reported additional analyses of soot, giving 12 ppm for 2,3,7,8-TCDF, 28 ppm for total tetra-CDF, 670 ppm for penta-CDF, 965 ppm for hexa-CDF, 460 ppm for hepta-CDF, and 40 ppm for octa-CDF (29).

PCDFs were observed in the residues from all the fires involving PCB-filled capacitors. At the transformer station in Norrtalje (in 1978), PCDF concentrations ranged from 1.1 ppm in an undamaged capacitor to 75 ppm in the fluid left inside an exploded capacitor, and 52 ppm on the outside of that same exploded capacitor (2). No PCDFs were detected on nearby trees, or in the blood samples taken from firefighters (2). Absolute confirmation of specific isomers was not possible (2, 3).

In the 1982 fire in Skovde which burned 21 PCB capacitors, PCDFs were found on the floor in and near the capacitor battery, on the wall of the capacitor room, and on a bench; 2,3,7,8-TCDF was found in all but the last instance (3). In the 1982 Stockholm fire, seven tetra-CDFs and 11 penta-CDFs were identified and quantified.

In the Yusho incident, PCDFs were found in the rice oil. The oil showed concentrations of 4 to 5 ppm after contamination with approximately 1000 ppm PCB. Tests of various PCB mixtures indicate PCDF contamination of 1 to 18 ppm; using 10 ppm PCDF in PCB and 1000 ppm PCB in rice oil, 0.01 ppm PCDF in rice oil would be expected. Actual findings of 5 ppm support the view that PCDF was formed from the PCB after prolonged heating (14, 15). In a laboratory simulation of the steam distillation process used, PCDFs were formed from the PCBs (32).

When Yusho broke out again in Taiwan, researchers reported 53 to 405 ppm PCB in the toxic oil and 0.18 to 1.68 ppm PCDF (18). From blood sample analyses, it also appeared that some oils, which were not available for analysis, had contained much higher levels of PCB (18). No identification of the PCDFs was reported.

Other Polychlorinated Organics

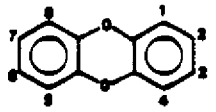
In addition to PCDDs and PCDFs, a number of other polychlorinated organics have been detected in samples from these incidents. PCQs, PCDEs, and PCTs were all detected in Yusho oil from Japan (16); PCQs and PCTs were detected in Yusho oil in Taiwan (18). PCBPs were detected in the residues of the Binghamton and Stockholm (1981) fires (3, 4). Evidence of polychlorinated pyrenes (PCPYs) and polychlorinated chrysenes (PCCYs) was found the 1982 Stockholm fire and in the Skovde fire; polychloroxanthenes (PCXs) were found in the St. Louis incident.

Section 3

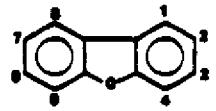
PCDD/PCDF/PCBP FORMATION

PHYSICAL AND CHEMICAL CHARACTERISTICS OF PCDDs AND PCDFs

Both the dibenzo-p-dioxin (or dioxin) and dibenzofuran compounds have a triple-ring structure consisting of two benzene rings connected by a third ring. In the dioxin (shown below), the connection is through a pair of oxygen atoms.



The middle ring of the dibenzofuran contains only one oxygen atom, as shown below.



Both compounds can have chlorine atoms at any or all of the numbered positions on the benzene rings. Theoretically, there are 75 possible different positional isomers of chlorinated dioxins, from mono- to octachloro, and 135 different chlorinated dibenzofuran isomers (Table 3-1 and Appendix A) (33, 34).

Very little published information is available on the chemical and physical properties of most of these compounds, particularly the polychlorinated dibenzofurans (PCDFs). The unsubstituted dioxin moiety is stable to at least 700°C and possibly to 1150°C (33). The 2,3,7,8-tetrachloro isomer is the most studied isomer due to its toxicity. This isomer is symmetrical across two planar axes. It is sparingly soluble in water and in most organic solvents; it is relatively inert to acids, bases, oxidation, and reduction, and is extremely lipophilic. In general, chemical stability and lipophilicity increase with increasing halogen content, although higher chlorinated isomers are more readily subject to attack by strong acids and

bases. 2,3,7,8-TCDD is a solid at room temperature, melting at 305°C. The octachloro isomer melts at 130°C.

Table 3-1

ISOMERS OF PCDFs AND PCDDs

Chlorine Substitution	Number of Isomers	
	PCDFs	PCDDs
Mono-	4	2
Di-	16	10
Tri-	28	14
Tetra-	38	22
Penta-	28	14
Hexa-	16	10
Hepta-	4	2
Octa-	1	1
	135	75

CHEMISTRY OF FORMATION OF PCDDs AND PCDFs

A summary of chemical pathways to PCDDs, PCDFs, and PCBPs is given in Figure 3-1.

Polychlorinated Dibenzo-p-Dioxins (PCDDs)

There are a limited number of direct precursors and reactions by which PCDDs can be formed. In order to be a dioxin precursor, a compound must meet two conditions (33):

- The compound must be an ortho-substituted benzene ring in which one of the substituents includes an oxygen atom directly attached to the ring.
- It must be possible for the two substituents, excluding the oxygen, to react with each other to form an independent compound.

An example of the ideal dioxin precursor is as follows:

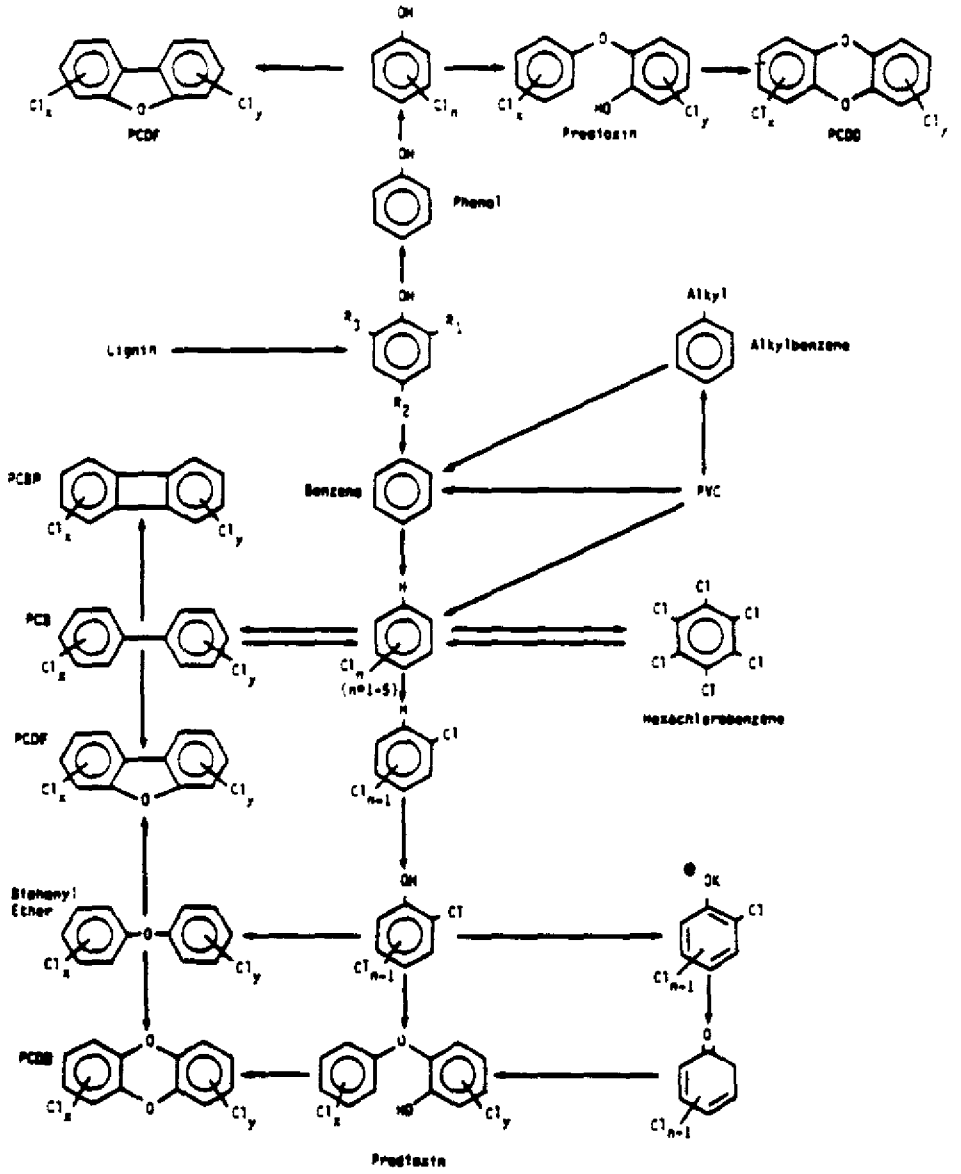


Figure 3-1. Chemical Pathways to PCDDs, PCDFs, and PCBPs

MONS 215219

A reaction between two of these molecules could yield a dioxin and 2 XY molecules, as follows:

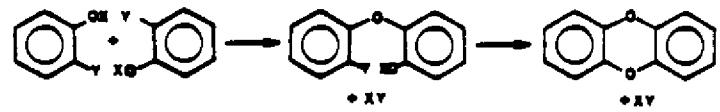
(rxn 1)

Although the number of direct dioxin precursors is relatively small because of the conditions set forth above, there are many indirect precursors, i.e., compounds which do not fit the model, but which can degrade or be transformed into direct precursors. This will be discussed in more detail later

Buser (25) and Rappe, et al. (35), have identified the following basic reaction for chlorinated dioxin formation:

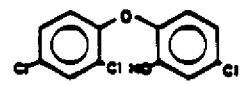
- Dimerization of chlorophenols and phenoxy acids (pyrolytic and photochemical) - A bimolecular reaction in which the relative ratio of isomers formed depends on the concentration of the chlorophenates; yields decrease in more dilute systems, such as contaminated mineral oils.
- Cyclization of predioxins (pyrolytic and photochemical) - Unimolecular, and thus concentration-independent.
- Dechlorination of higher chlorinated PCDDs - Unimolecular, and thus concentration-independent.
- Direct chlorination of dibenzo-p-dioxins.

The dimerization reaction (rxn 1) basically follows a two-step mechanism (33):



(rxn 2)

The intermediate diphenyl ether compound is called a predioxin. Typically, a predioxin will either cyclize to dioxin or break down to the precursors. For example, in one test, Irgasan DP300, a compound with a predioxin configuration



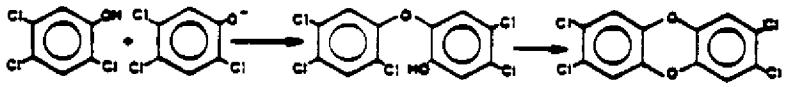
was heated to 980°C (36). The reaction yielded both dioxins and precursors. Because of such competing reactions, dioxins are seldom found in more than trace quantities. It has been shown that the higher chlorinated predioxins are amenable to self-condensation, while the lower chlorinated predioxins apparently do not readily cyclize further (37). For instance, in one test, irradiation of 2,4-dichlorophenol yielded a predioxin, but no further reaction (33).

Temperatures for dioxin formation by dimerization/cyclization have been reported at values from 180°C to 620°C (33). Sandermann, et al. (cited by Choudhry and Hutzinger) (38), obtained octachlorodioxin (OCDD) (6 percent) and hexachlorobenzene (51 percent) upon heating 100 g of pentachlorophenol in a closed tube at 300°C. Esposito, et al. (33), reported quantitative yields of OCDD from heating the sodium salt of pentachlorophenol at 360°C. Ahling and Lindskog (39) found that combustion of tri- and tetrachlorophenols at 500° to 600°C can yield PCDD. Duckett (40) reported temperatures for dioxin formation from pentachlorophenol ranging from 310° to 370°C.

However, the formation of dioxins from chlorophenols is exothermic, although there are no published data on the amount of heat released (41, 42). This fact, combined with the need for heating to initiate the reaction, suggests that one or more of the reaction steps in the dioxin formation sequence has a high activation energy. The high activation energy contributes directly to the low yields. In addition, the boiling points of many dioxin precursors are lower than the reaction temperature range. Thus, there is a loss of precursor molecules before reactions can occur (40). Most laboratory syntheses of dioxins rely on closed reaction vessels or pressure to keep precursors in the liquid state.

Another major source of penta- and tetra-CDD isomers is photochemical dechlorination of higher isomers, mainly OCDD (43). The principal toxic isomers, 1,2,3,7,8-pentachloro and 2,3,7,8-tetrachloro, are not as readily formed as other penta- and tetrachloro isomers. The major hepta-CDD formed is the 1,2,3,4,6,7,9-substituted isomer, indicating a preferential loss of lateral (2,3,7-, or 8-) chlorine atoms (44, 45). The major hexa-CDD formed is either 1,2,4,6,7,9- or 1,2,4,6,8,9-hexa-CDD (44). The major penta-CDD is expected to be the 1,2,4,6,9-substituted isomer, and the major tetra-CDD is 1,4,6,9-tetra-CDD (44). These reaction products show a preferential removal of the lateral chlorine atoms. Lower isomers (di- and trichloro) may be formed by irradiation, but they decompose via the same route, and thus do not accumulate (46).

Other known sources include condensation of catechols with polychlorobenzenes or chloronitrobenzenes (33) (Figure 2-1). Pyrolysis (620°C) of chlorobenzenes in the presence of air yields PCDDs (33, 47) Since chlorobenzenes do not fit the conditions set down earlier for dioxin precursors, Buser (25, 49) has postulated that the alkaline hydrolysis of chlorobenzene to PCDD involves a two-step condensation process from ortho-chlorinated phenoxy anions via a polychlorinated 2-phenoxy phenate.



(rxn 3)

This dimerization is exothermic. Below approximately 180°C, usually very minor amounts of PCDDs are formed (<1 ppm), but significant quantities (1 to 10 percent yields) result at temperatures over 180°C (25).

Dioxins have also been detected in residues from a variety of combustion processes, including municipal incineration, the burning of fossil fuels, wood in wood stoves, and cigarettes (50). Precursors of dibenzo-p-dioxins are also known to be formed after simply adding hyorogen chloride to the flame of a Bunsen burner (50). Most investigations have been centered on municipal incinerators and industrial heating plants. Olie, et al. (50), for example, analyzed fly ash samples from a municipal incinerator in Netherlands, and found average PCDD concentrations of 2056 ng/g. Hexachloro isomers predominated, followed by heptachloro isomers. Liberti, et al. (52), found total concentrations of PCDDs and PCDFs on the order of 1300 ppb in Italian municipal incinerator fly ash. They suggested phenolics plus chlorine or HCl as precursors. Buser, et al. (53), also detected PCDDs in Swiss municipal fly ash. The major isomers detected were the same as those formed in the pyrolytic dimerization of the more common chlorophenates, namely the 2,4-di-, 2,4,6-tri-, 2,3,4,6-tetra-, and pentachlorophenates.

The mechanism(s) of PCDD formation in fly ash is presently unknown. Two suggested sources are:

- Condensation of chlorophenols.
- Thermal synthesis from any organic materials and inorganic chloride (the "trace chemistries of fire" hypothesis) (54).

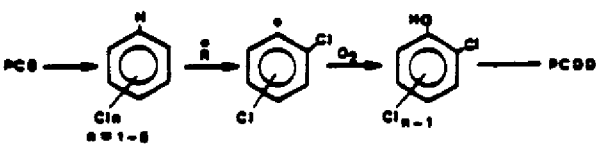
However, even ideal PCDD precursors, such as polychlorophenols, show very low PCDD conversion efficiencies. Thus, although every chlorinated organic compound or

organic compound/inorganic chlorine mix might be regarded as a potential precursor, the range of likely precursors for formation of detectable quantities of PCDDs is probably not that broad (55). Consequently, it is unlikely that organic material/ inorganic chloride reactions will produce PCDDs.

The high-temperature air oxidation of coal in the presence of chlorine, HCl, or NaCl has been shown to yield PCDDs (47) However, coal is a commercial source of benzene and cumene (a phenol raw material, Figure 2-1), so that formation of chlorinated benzenes and phenols is a probable intermediate step. Thermal decomposition of lignin (Figure 2-1) and polyvinyl chloride (typical constituents of municipal refuse) can yield phenols, cresols (Figure 2-1), catechols, benzene, chlorobenzenes, HCl, etc. (46, 56). Thus, rather than just any organic material serving as a potential precursor, it seems more likely that those compounds which can be pyrolyzed to benzene, chlorobenzene, phenols, catechols, and/or inorganic chlorine are more probable indirect precursors.

The mechanism(s) by which PCDDs are formed during combustion is still not well understood; thus, the effects of combustion temperature, fuel characteristics, catalysis, and other parameters have not been determined (57). Townsend (58) has suggested that dioxins created within a combustion source probably have a composition which reflects the chemical conditions within the source. Upon emission, a chemical equilibrium process takes place, yielding isomer ratios reflecting the chemical conditions of the atmosphere. However, this theory has not been generally accepted.

There has been some question regarding the role of PCBs in PCDD formation. In 1981, an electrical fire in the State Office Building in Binghamton, New York, resulted in the pyrolysis of PCB fluid from a transformer (4, 5, 6). Soot from this fire contained PCDFs and PCDDs. PCBs do not fit the precursor model discussed earlier, and PCDDs would not be expected to form readily in a mixture of biphenyl compounds (59). Conceivably, PCBs could be oxidized to polychlorinated hydroxybiphenyls which could, in turn, cyclize to form dioxins (60). Another reaction that has been suggested is (47):



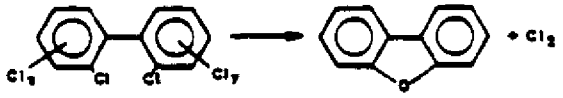
(rxn 4)

However, neither mechanism quite fits the thermal decomposition pattern for PCBs.

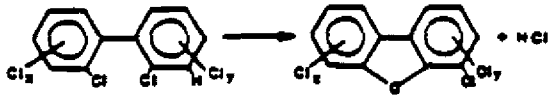
The PCB fluid at Binghamton was a mixture of 65 percent Aroclor 1254 and 35 percent chlorobenzenes. Rappe, et al. (61), have suggested that pyrolysis of the chlorobenzenes is a more likely source of the PCDDs than PCBs. This reaction was discussed earlier.

Polychlorinated Dibenzofurans (PCDFs)

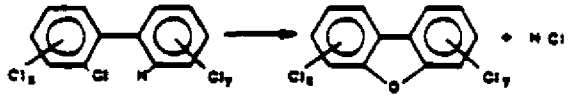
While PCDFs and PCDDs can share some of the same precursors, PCBs are the most common source of PCDFs. Buser and Rappe (34) have identified four basic mechanisms of PCDF formation from PCBs:



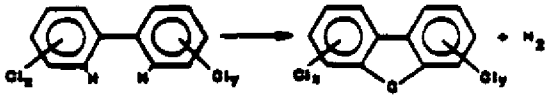
(rxn 5)



(rxn 6)



(rxn 7)



(rxn 8)

Reaction 4 involves the loss of the ortho-Cl. Reaction 5 is a loss of HCl involving a 2,3-chlorine shift. Reaction 6 has a loss of ortho-HCl. Reaction 7 involves a loss of ortho-H.

PCDFs have been identified as trace contaminants at the ppm level in a number of American, European, and Japanese commercial PCB mixtures (13, 53, 62, 63).

Concentrations and isomer ratios vary with the type of PCB and the country of origin, probably as a result of differences in manufacturing conditions (Table 3-2). For instance, Bowes, et al. (63), detected PCDFs in American-manufactured Aroclor 1260 and 1248 at total concentrations of 0.8 and 2.0 ppm, respectively; none were detected in a single sample of Aroclor 1016. The total quantities of PCDFs in German Clophen A-60 and French Phenoclor DP-6 were estimated to be 8.4 and 13.6 ppm, respectively (62). PCDFs have also been detected in several Japanese-made PCBs at concentrations from 1 to 20 ppm, with the highest concentration in a sample of Kanechlor KC-400 (11, 14).

Table 3-2

LEVELS (ug/g) OF PCDFs IN COMMERCIAL PCBs

Sample	Tri-	Tetra-	Penta-	Hexa-	Hepta-	Total	Reference
Aroclor 1248 (1969)	-	0.5	1.2	0.3	-	2.0	63
Aroclor 1242	-	0.07	0.03	0.003	-	0.15	65
Aroclor 1242	-	2.3	2.2	N.D.*	-	4.5	15
Aroclor 1243	0.1	0.25	0.7	0.81	-	1.9	64
Aroclor 1254 (1969)	-	0.1	0.2	1.4	-	1.7	63
Aroclor 1254 (1970)	-	0.2	0.4	0.9	-	1.5	63
Aroclor 1254	-	0.02	0.2	0.4-0.6	-	0.8	66
Aroclor 1254 (KK 602)	-	0.05	0.1	0.02	-	0.2	64
Aroclor 1254	-	0.1	3.6	1.9	-	5.6	15
Aroclor 1260	0.06	0.3	1.0	1.10	1.35	3.8	64
Aroclor 1260 (1969)	-	0.1	0.4	0.5	-	1.0	63
Aroclor 1260	-	0.8	0.9	0.5	-	2.2	15
Aroclor 1260 (AK 3)	-	0.2	0.3	0.3	-	0.8	63
Aroclor 1016 (1972)	-	N.D.	N.D.	N.D.	-	-	63
Clophen A60	-	1.4	5.0	2.2	-	8.4	63
Clophen T64	0.1	0.3	1.73	2.45	0.82	5.4	64
Phenoclor DP-6	-	0.7	10.0	2.9	-	13.6	63
Prodelec 3010	0.41	1.08**	0.35	0.07	-	2.0	64
Kanechlor 400	-	-	-	-	-	ca. 20.0	66
Mitsubishi (used)	2.13	4.00	3.30	0.53	-	10.0	64

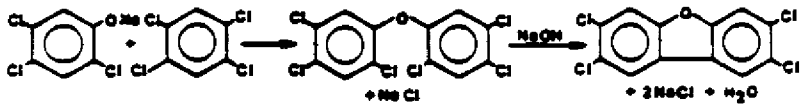
* N.D. = None Detected.
** Major isomer 2,3,7,8-tetra-CDF.

PCDFs can also form during PCB use. The best known example of this is the "Yusho" incident in Japan, which is discussed in more detail in Section 2. The PCBs, which

had leaked from a heat exchanger, had PCDF/PCB ratios 200 to 250 times higher than that of the original Kanechlor KC-400 (11, 64). Furthermore, the PCDFs in the Yusho oil contained the same isomers as a sample of used PCBs (10). This suggested that most of the PCDFs had been formed in service (62). Similar PCDF isomer ratios have been detected in other used Japanese PCBs (43).

In an experiment designed to duplicate the Yusho oil incident, Morita, et al. (67), heated PCBs in air for 1 week. PCDFs were formed at temperatures over 270°C, reaching a maximum level at 300°C, and then declining at 330°C, suggesting decomposition Primarily, di- and trichloro dibenzofurans were formed in this experiment, while tetra- and pentachloro isomers were found in the Yusho oil. Jansson and Sundstrom (2) showed that, at 550° to 650°C, 0.5 to 2.8 percent of PCBs converted to PCDF, while no net formation was observed above 700°C.

Other sources of PCDF include condensation of chlorophenates and chlorobenzenes, photochemical degradation of higher chlorinated isomers, cyclization of polychlorinated diphenylethers and polychlorobenzenes, and direct chlorination of dibenzofurans (45). Buser (45) showed that octachloro isomers could be photochemically dechlorinated to tetra- and penta-CDFs. The preferential positions for photochemical loss of chlorine in the dibenzofuran system have not been studied. In their discussion, Choudhry, et al. (47), noted that both chlorinated diphenylethers and chlorobenzenes will yield PCDFs and PCDDs when heated to 600° to 620°C. Chlorophenols and chlorobenzenes can form PCDFs via an intermediate polychlorinated diphenylether (PCDPE) (25, 33).



(rxn 9)

PCDPEs usually occur as contaminants in chlorophenols, and are known to form PCDFs by thermal, photochemical, or metal-catalyzed reaction (68). PCDPEs and PCDFs probably form early in the reaction when large concentrations of chlorobenzenes are still present, whereas PCDDs are formed toward the end of the reaction (68).

Fires in electrical equipment containing PCBs have resulted in PCDF formation. Jansson and Sundstrom (2) investigated the aftermath of a 1978 capacitor fire in Sweden. Whereas the original PCB contained 1.1 to 1.3 ug PCDF/g (amounts of mono-,

di-, tri-, and tetra- isomers about equal), PCB samples from inside the exploded capacitors averaged 81 ug PCDF/g PCB (approximately one-half of PCDF di- isomer, one-third mono-, and the rest tri-, with a trace of tetra-), and samples from the inside surfaces contained 45 to 107 ug PCDF/g PCB (predominantly di- and mono- isomers with some tri- and a trace of tetra-). Soot from a 1977 transformer fire in Toronto contained 5 ug PCDF/g, while the original PCB contained 0.05 ug PCDF/g (2). During the course of the 1981 Binghamton State Office Building electrical fire, the secondary bushing of a 1100-gallon (4160-liter) transformer cracked, releasing about 180 to 200 (680 to 760 liters) gallons of transformer fluid (65 percent Aroclor 1254 PCB, and 35 percent tri- and tetrachlorobenzene) (69, 70). Chemical-contaminated soot was spread throughout the building by the ventilation system. Two soot composite samples collected from the stairwells contained 120 and 270 ppm 2,3,7,8-TCDF (70). Analyses of soot samples by Stalling and Rappe/Buser showed PCDF/2,3,7,8-TCDF levels of 756 ppm/3.7 ppm and 2160 ppm/ 12 ppm, respectively (70).

In September 1982, a 500-unit capacitor battery at a steel mill in Surahammar, Sweden, was ignited by 10 tons (9090 kg) of molten steel. The capacitors were filled with PCBs (2 tons [1818 kg]) and mineral oil (3 tons [2727 kg]). The fire burned for 2 to 3 hours, and filled the entire building with smoke. Table 3-3 presents the results of analyses of wipe samples.

There have been several other capacitor fires after which PCDFs were found, such as those at a paper mill in Imatra, Finland (August 1982), and Volvo in Skovde, Sweden. Table 3-4 presents a comparison of the gross PCDF from several incidents. Figure 3-2 shows very similar TCDF isomer distributions from these same incidents.

PCDFs have also been detected in fly ash samples from municipal incinerators and industrial heating facilities. Incinerator fly ash from the Netherlands averaged 1309 ng PCDF/g (51). Incinerator fly ash from Italy averaged about 1300 ppb of combined PCDD and PCDF (52). Fly ash from an industrial heating facility and municipal incinerator in Switzerland contained 0.3 and 0.1 ppm PCDF, respectively (53). Thus far, the exact source of the PCDFs in fly ash is unknown. Buser, et al. (53), pyrolyzed commercial PCBs in quartz tubes, and produced the same relative PCDF isomer ratios that were detected in the Swiss fly ash, suggesting that PCBs were the source of the PCDFs. Other researchers have suggested that the similar isomer distribution is a function of the reaction surface (quartz tubes are SiO_2, and fly ash is generally over 50 percent SiO_2), and do not necessarily indicate the same precursors (72).

Table 3-3

PCDFs FOUND AT SURAHAMMAR, SWEDEN (ng/m^2)*

Sample Location	TCDF	2,3,7,8 TCDF	Cl_5	Cl_6	Cl_7	Cl_8
Capacitor Room (Sample 1)	4000	875	3300	1800	1500	300
Capacitor Room (Sample 2)	1100	365	1250	940	625	145
N.E. Corner, 10 m height	1250	300	355	150	65	13
S.E. Corner, 10 m height	480	120	210	140	60	30
N.E. Corner, floor	100	25	27	15	5	2
S.E. Corner, floor	90	22	25	17	17	4
10 m Outside, downwind	<250	<25	<25 (5)	<60 (10)	58**	17**
300 m Outside, downwind	<250	<25	<25 (5)	<60 (10)	<30	<12
After Cleaning (Sample 1)	<20	<4	<10	<12	<15	2**
After Cleaning (Sample 2)	<40	<8	<20	<20	<30	6**

Source: C. Rappe, Personal Communication.

* Wipe tests.
** Possibly due to Cl_9-Cl_{10} PCB in casting sand.

Table 3-4

PCDF LEVELS IN PCB FIRES

Site	PCDF Levels	PCBP Levels*
Skoevde (foundry, well-ventilated)	0.01 ug/m^2	None
Skoevde (basement capacitor room)	0.8 ug/m^2	None
Danniken, Sweden	1.5 ug/m^2	1.5 ug/m^2
Surahammar, Sweden	1-10 ug/m^2	Less than PCDFs
Imatra, Finland	1-3 ug/m^2	Less than PCDFs
Imatra, Finland	1.5 ppm	
Binghamton, New York	2160 ppm	Less than PCDFs

* Quantification not possible due to the lack of standards.

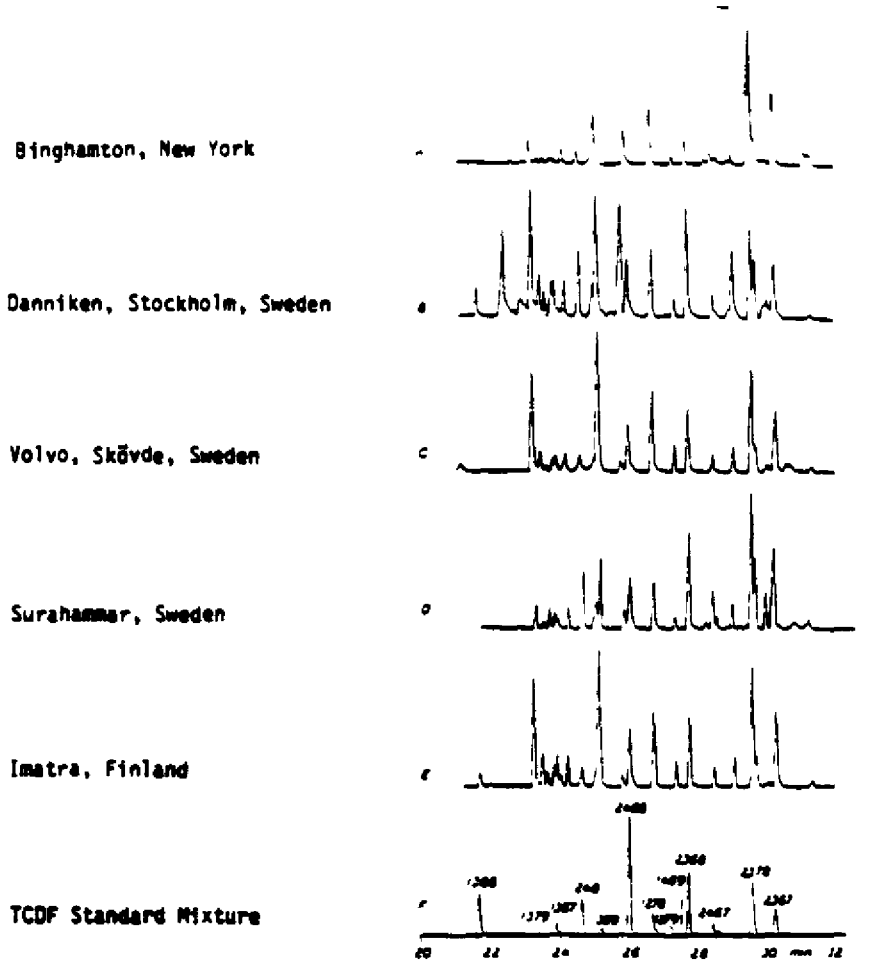


Figure 3-2. TCDF Isomer Distributions at Five PCB Fires

TRANSFORMER/CAPACITOR CHEMISTRY

Askarel transformers are those transformers designed and manufactured to contain a liquid fire-resistant, electrically insulating coolant (askarel), consisting of 60 percent (600,000 ppm) to 100 percent (1,000,000 ppm) commercial PCB products (66), and up to 40 percent trichlorobenzene (1). The term "PCB transformer," as defined in 40 CFR, Sec. 761.3(y), includes all askarel transformers (as defined above), plus transformers filled with mineral oil but contaminated with PCBs at concentrations of 500 ppm or greater.

Most PCB-filled electrical equipment is designed to operate at temperatures of 100° to 130°C, and transformer "hot spots" are typically no greater than 135°C (66). All units may contain air, but most newer ones have an inert atmosphere, usually nitrogen. Askarel units are sealed, but some seals can deteriorate with time, permitting the entry of air and water. Hydrochloric acid can be generated, leading to internal corrosion. Water can cause arcing. Internal transformer malfunctions can cause bushings and winding connections to become white hot. During these occasional short-circuit episodes, temperatures reach 200° to 250°C for a few minutes. In cases of an arc where the transformer fails, maximum temperatures may reach 1000°C; these failures occur within fractions of a second (66). Capacitors typically operate below 100°C. In those capacitor failures resulting in rupture, heating and internal arcing may have occurred prior to rupture.

Samples of used oil taken from two capacitors (Prodelec 3010) have been analyzed for PCDFs; no apparent increase was detected (see Table 3-2) (19, 64). The formation of PCDFs when capacitor oils are exposed to sufficient oxygen and high temperatures was investigated by conducting laboratory tests with Prodelec 3010 (19). The data indicated that both sparking and soldering did not result in an increase in the concentration of PCDFs; weldings, however, resulted in a 1.4- to 5-fold increase in PCDFs (19).

Samples of used askarel taken from transformers manufactured in the United States and Canada have also been analyzed for PCDF concentrations. For instance, both bulk samples of used askarel and air in a transformer maintenance workplace were analyzed; results showed trace TCDF concentrations (0.013 to 0.116 ppm) in the fluids, and no detectable concentrations in the air (73). Two samples of used askarel were also analyzed by the National Institute of Environmental Health Sciences (74). Fluid A, obtained from a transformer of early manufacture but unknown extent of use, contained 0.7 ppm PCDF (66). Fluid B, a bulk sample of used askarel obtained from a

transformer maintenance operation (66), contained 4.7 ppm PCDF (74). The PCDFs found in Fluid B consisted primarily of octa-CDF (4.5 ppm), and no detectable tetra-TCDF.

In 1979, Chittim and coworkers (75) investigated the formation of TCDFs in transformer askarels, using gas chromatography with a packed column. They reported TCDF concentrations ranging from approximately 0.004 to 4.7 ppm in used askarels (Table 3-5). The amount of PCDDs, if detected, was usually less than 5 percent of the TCDF concentrations (75). Small amounts of terphenyl, quaterphenyl, and chlorinated terphenyl were also detected in some of the askarel samples, but they were not quantified. The concentrations of TCDF in transformer askarels were found to increase with time in service. Furthermore, the apparent changes in TCDF concentrations caused by discharging or arcing were found to be negligible, most likely because of the short duration of the sparks or events causing failure. It should be noted that the authors themselves acknowledged the limitations of the applied analytical method (i.e., packed chromatographic column).

In 1982, the General Electric Company (GE) submitted a sample of used askarel (obtained from the Canadian General Electric Company) to both a university-affiliated and an industrial laboratory for PCDF analysis. Using high-resolution gas chromatography/mass spectroscopy (GC/MS), the industrial laboratory found PCDFs on the order of 1.0 ppm or lower (66). The university-affiliated laboratory found no PCDFs at detection limits of 10.0 ppm for tetra- and pentachloro isomers, and 1.0 ppm for hexa-, hepta-, and octachloro isomers (66).

Subsequently, GE submitted four additional samples of used askarel to the same industrial laboratory for PCDF analysis (66). These samples had been obtained from in-service distribution transformers at a GE transformer manufacturing facility in the United States during the course of normal maintenance activities. The results of these analyses, presented in Table 3-6, show PCDF concentration ranges from 0.62 to 1.86 ppm in 40-year-old askarels; these PCDF concentrations are comparable to the PCDF concentrations reported for unused Aroclors.

The formation of PCDFs when askarels are exposed to sufficient oxygen and high temperatures was demonstrated by conducting laboratory-scale pyrolysis at 500°C of two PCB mixtures (Aroclor 1254 and Aroclor 1016) and three askarels (Chlorextol, Pyranol, and Inerteen) (75). The results of these tests (Table 3-7) indicated the formation of high concentrations of PCDFs. Aroclor 1016 generated significantly

Table 3-5

PCBz* AND TCDF CONCENTRATIONS (ppm)
IN USED ASKARELS (75)

Sample**	Installation	1,3,5 TCBz	1,2,4 TCBz	1,2,3 TCBz	1,2,3,5- and 1,2,4,5-TCBz	1,2,3,4 TCBz/#	1,2,3,4,5 PCBz	PCBz/PCB	Aroclor	TCDF
Group 1										
1-1	1957	tr	26.4	6.8	1.0	14.4	2.4	51.0/49.0	1260	0.780
1-2	1961	tr	28.4	6.6	tr	15.2	3.4	53.8/48.4	1260	1.110
1-3	1963	tr	27.8	6.2	tr	14.0	2.8	50.6/49.4	1260	1.320
1-4	1963	tr	28.6	6.4	tr	14.4	2.5	52.4/47.6	1260	0.790
1-5	1963	tr	28.4	8.0	tr	2.2	tr	38.6/61.4	1260	0.350
1-6	1965	tr	28.0	7.6	tr	1.6	ND	37.2/62.8	1260	0.290
1-7	1970	tr	22.6	7.0	tr	tr	ND	29.6/70.4	1254	0.550
1-8	1970	tr	21.2	6.8	tr	tr	ND	28.0/72.0	1254	0.470
1-9	1971	tr	21.2	7.8	tr	tr	ND	29.0/71.0	1254	0.290
1-10	1974	ND	21.6	6.8	ND	ND	ND	28.4/71.6	1254	0.051
1-11	New	tr	20.8	9.8	tr	tr	ND	30.6/69.4	1254	0.041
Group 2										
2-1	1967	ND	21.4	9.6	tr	14.8	3.4	45.2/54.8	1260	0.030
2-2	1967	tr	26.2	7.2	tr	14.8	3.2	44.3/55.7	1260	0.004
2-3	1967	tr	26.0	7.0	tr	14.4	3.2	50.6/49.4	1260	0.007
2-4	1967	tr	28.4	6.4	tr	13.4	2.8	49.0/51.0	1260	0.010
2-5	1965	tr	27.6	7.8	tr	tr	tr	35.4/64.6	1260	0.176
2-6	1965	tr	27.0	5.8	tr	13.6	2.6	49.0/51.0	1260	0.435
2-7	1965	tr	27.8	6.6	tr	1.0	tr	35.4/64.6	1260	0.110
2-8	1965	tr	28.4	8.2	tr	tr	tr	36.6/63.4	1260	0.141
Group 3										
3-1	1967	ND	21.4	5.6	tr	14.8	3.4	45.2/54.8	1260	0.419
3-2	1967	tr	26.0	7.0	tr	14.4	3.2	50.8/49.4	1260	0.341
3-3	1967	tr	26.2	8.0	tr	tr	tr	36.2/63.8	1260	0.444
3-4	1967	tr	27.4	7.8	tr	tr	tr	35.2/64.8	1260	0.426
3-5	1967	tr	28.0	7.8	tr	tr	tr	32.8/67.2	1260	0.1
3-6	1967	tr	28.0	7.8	tr	tr	tr	32.8/67.2	1260	0.1
Group 4										
4-1	1967	ND	24.8	7.2	tr	14.2	3.0	49.2/50.8	1260	4.7
4-2	1967	ND	25.2	5.6	tr	14.0	2.8	48.4/51.6	1260	2.2
4-3	1965	ND	27.0	5.2	tr	14.4	3.6	50.2/49.8	1260	0.4
4-4	1973	ND	27.4	5.2	tr	14.8	4.0	51.4/48.6	1260	0.4
4-5	New	tr	35.4	13.4	tr	10.0	ND	58.8/41.2	1254	0.0
4-6	New	ND	20.2	10.0	tr	ND	ND	30.2/69.8	1254	0.0
4-7	New	tr	20.8	9.8	tr	tr	ND	30.6/69.4	1254	0.0

* PCBz = polychlorinated benzenes

** Group 1: (1-1 through 1-11) - variable time-in-service (FP).

Group 2: (2-1 through 2-4) - CGE transformers; variable power/volume ratio (see Appendix C)
(2-5 through 2-8) - FP transformers; variable power/volume ratio (see Appendix C)

Group 3: Variable fluid type - 3-1 and 3-2 Pyranol (CGE)
3-3 and 3-4 Chlorextol (FP)
3-5 and 3-6 Inerteen (W).

Group 4: 4-1 and 4-2 - "very old" transformers (FP); large variation in power/volume ratio (see Appendix C).
4-3 and 4-4 - transformers which had failed in service (AD, WA).
4-5, 4-6, and 4-7 - new Pyranol (CGE), Inerteen (W), and Chlorextol (FP)

Manufacturer: (Fluid type)		
	CGE - Canadian General Electric	(Pyranol)
	FP - Ferranti-Packard	(Chlorextol)
	W - Westinghouse	(Inerteen)
	AD - Brown Boveri	(Pyroclor)
	WA - Foster Electric	(Inerteen)

Chloranaegan (76).

Table 3-6

CONCENTRATION OF PCDFs IN ASKAREL TRANSFORMER FLUIDS FROM IN-SERVICE DISTRIBUTION TRANSFORMERS AT GE TRANSFORMER MANUFACTURING FACILITY (75)

Sample Number	Type of Transformer	Date Installed	Total PCDF (ppm)	% PCB
3	Single phase - 100 kVA 2300 vt -- 115/230 115 gallons (435 liters) fluid	1940	1.1	56.4
23	3 phase - 1250 kVA 1380 vt -- 480Y/277 490 gallons (1855 liters) fluid	1947	0.62-0.82	66.2
44	2 phase - 375 kVA 2300 vt -- 550 31.5 gallons (119 liters)	1938	1.84-1.86	60.1
45	Single phase - 100 kVA 2300 vt -- 120/240 42 gallons (159 liters)	1939	0.64-0.86	66.8
"Pure Aroclor 1254" (control)			0.62-0.82	

MONS 215233

Table 3-7

PYROLYSIS OF PCBs AND ASKARELS AT 500°C (7S)

PCB or Askarel	Mass Pyrolyzed (mg)	Position of Sample*	Mass of Tetrachlorodibenzofurans Formed: Actual Mass (ug)	Mass of Tetrachlorodibenzofurans Formed: Per Gram of Starting Material (ug/g)	Mass of Tetrachlorodibenzofurans Formed: Total (Q+C)	Mass of Tetrachlorodibenzofurans Formed: Average of Both Runs (ug/g)
Aroclor 1254	25.0	Q	2.20	88.0		
		C	1.33	53.2	141.2	
						169.3
	40.0	Q	4.90	122.5		
		C	3.03	75.8	197.3	
Aroclor 1016	25.0	Q	0.038	1.52		
		C	0.027	1.08	2.60	
						2.53
	40.0	Q	0.072	1.80		
		C	0.026	0.65	2.45	
Pyranol	25.0	Q	0.53	21.2		
		C	0.53	21.2	42.4	
						NA
	0.25	Q	0.048	191.7		
		C	0.074	297.0	488.8	
Inerteen	0.25	Q	0.030	118.3		
		C	0.030	121.9	240.2	
						193.1
	0.25	Q	0.033	133.7		
		C	0.003	12.2	145.9	
Chlorextol	0.25	Q	0.023	93.5		
		C	0.025	100.2	193.7	
						221.1
	0.25	Q	0.021	82.8		
		C	0.041	165.7	248.5	

* Sampling points: Q = immediately after the reaction tube; C = a cold trap further along in the reaction apparatus.

less PCDF than Aroclor 1254 when pyrolyzed. These results were to be expected since Aroclor 1016 has lower chlorine content, and thus does not contain as much of the higher chlorinated biphenyls (i.e., penta- or hexachloro), which are the necessary precursors of TCDFs.

As discussed above, PCB conversion to PCDF or chlorobenzene conversion to PCDD and PCDF might be possible in a malfunctioning transformer or capacitor, provided that there is sufficient time at optimum temperature and/or oxygen conditions for their formation. Ordinary working conditions do not appear conducive to PCDD/PCDF formation (66).

As described in Section 2, electrical equipment PCB fires may begin internally or externally. If they begin internally in an airless transformer or capacitor (with either no head space or an inert atmosphere), the early stages are caused by electrical energy in interval arcing. Under these conditions, the major combustion products are PCBPs; smaller amounts of PCDFs, PCPYs, and PCCYs are also found (C. Rappe, Personal Communication). PCBPs were identified for the first time in the aftermath of a 1981 capacitor explosion in Stockholm, Sweden (3).

If the equipment explodes or cracks and the PCBs are exposed to air, higher concentrations of PCDFs will form. If the fire begins externally, PCDFs are the major combustion product accompanied by PCPYs and PCCYs; no PCBPs will form. PCDDs will form only if chlorobenzenes/chlorophenols are present in the PCB or askarel. Details of detected combustion products after different electrical equipment incidents are given in Section 2.

Finally, it should also be noted that most of the research on PCB/askarel conversion to PCDD, PCDF, and other chlorinated species has been performed using pure PCBs or askarel formulations containing at least 50 percent PCBs. Very little research has been conducted on dilute PCB systems, such as contaminated mineral oils. Consequently, there is little data available on the effects of dilution on PCB chemistry.

Section 4

CHEMICAL AND BIOLOGICAL DEGRADATION

Very little research has been done on degradation mechanisms of dioxins and dibenzofurans. This is particularly true of the chlorinated dibenzofurans; there is almost no information published on degradation. A number of researchers have speculated on degradation pathways, but little actual research has been conducted. Consequently, the discussion must center primarily on the chlorinated dioxins.

CHEMICAL DEGRADATION

Ozonation

Ozone bubbled through a suspension of 2,3,7,8-TCDD in water and carbon tetrachloride for 50 hours achieved 97 percent degradation (33). The dioxin was apparently suspended as an aerosol combined with the CCl_4, which facilitated the ozone attack. Although not tested with dioxins, the combination of ozone and ultraviolet irradiation has been successfully used to degrade pentachlorophenol and DDT (33). The UV activates the molecules to a highly energetic state, making them more susceptible to ozone attack.

Chloroiodide Destruction

Recently, Botre, et al. (77), treated contaminated soil from Seveso, Italy, with several chloroiodides (a class of compounds derived from quaternary ammonium salt surfactant). In laboratory tests on residues from the vacuum evaporation of 2,3,7,8-TCDD in benzene, alkyldimethylbenzylammonium (benzalkonium) chloroiodide achieved 71 percent 2,3,7,8-TCDD degradation. The use of 1-hexadecylpyridinium (cetylpyridinium) achieved 92 percent degradation. In tests on actual soil samples, benzalkonium chloride achieved only about 14 percent degradation after 24 hours. When benzalkonium chloroiodide was added, an additional 38 percent was degraded. The chloroiodides apparently cleave the ether linkage.

Alkaline Dehydrochlorination

Alkaline dehydrochlorination is a generic term for several new chemical processes that have been developed to treat and dispose of hazardous chlorinated hydrocarbon

wastes. However, only the process developed by the Vertac Chemical Company of Memphis, Tennessee, has been tested on TCDD and other chlorinated dioxins (78).

The Vertac process involves the dehalogenation of chlorinated dioxins with anhydrous alkali metal salts of polyhydroxy alcohols at atmospheric pressure, or by reacting PCDDs, an alcohol, and a water solution of an alkali metal hydroxide. Vertac claims that 2,3,7,8-TCDD and chlorinated dioxins are reduced to essentially zero (78).

Experiments have been conducted by Vertac using 2,4,5-TCPh still bottoms, containing up to 250 ppm TCDD. A stoichiometric excess (based on the halogen content) of alkali metal alcoholates of C_{4-6}-alcohols, C_{2-5}-alkanepolyols, and monoalkyl ethers was used. The reaction took place at a temperature of 140°C to 220°C for a time sufficient to convert the organic halogen into inorganic halides. The reaction product was essentially free of 2,3,7,8-TCDD (ppt range) and other chlorinated dioxins (i.e., more than 99.95 percent destruction) (79).

Catalytic Reduction with Iron Chlorides

In 1972, experiments were conducted to investigate the potential for separation of esters of 2,4-D from 2,4,5-T in Agent Orange by fractionation (78). Overheads were found to contain 93 percent 2,4-D ester, whereas bottoms comprised 93 percent 2,4,5-T ester. It was not possible to determine TCDD closure by materials balance, as the stainless packing and steel column initiated some catalytic destruction of the TCDD. The bottoms fraction (2,4,5-T) contained more than 93 percent of the total TCDD, while the overhead fraction (2,4-D) contained 0.15 ppm TCDD. These experiments demonstrated that an effective and efficient separation of the esters was feasible (78).

Catalytic Oxidation with Ruthenium Tetroxide

Ruthenium tetroxide has been successfully employed in the laboratory to destroy 2,7-DCDD and TCDD (77). Reaction kinetics were studied using solutions containing 70 to 72 ppm PCDD in carbon tetrachloride and 2 to 4 ppm ruthenium tetroxide as a function of temperature. The results indicated a 2.4-fold increase in the rate constant per 10°C increase in temperature. At 70°C, the half-life of TCDD was found to be less than 15 minutes, and that of 2,7-DCDD, approximately 10 minutes.

Micellar Catalysis with Chloroiodides

The most promising results have been obtained with benzalkonium dichloroiodide and cetylpyridinium dichloroiodide (77). The low solubilities of these compounds were increased by the use of micellar solutions of the same surfactants. TCDD degradation products included chlorophenols, phenols, and 2-phenoxy-chlorophenol. These findings indicated that micellar catalysis (which is carried out in the absence of harsh acidic conditions, at ambient temperature, and without the use of light energy) results in a cleavage of the ether link.

Chlorolysis

The chlorolysis process operates with chlorine gas as the oxidant at a temperature of 600°C and a pressure of 2500 psig (170 atm). The Hoechst-Uhde Corporation, under contract to EPA, conducted an experimental study to assess the feasibility of the chlorolysis process to convert typical chlorinated solvent wastes and Agent Orange to useful products such as carbonyl chloride, hydrogen chloride, and carbon tetrachloride (78). The primary interest of the study was the destruction of the TCDD. The pretreated military defoliant initially contained 14 to 18 ppm TCDD, whereas the carbon tetrachloride product contained no measurable TCDD at a detection limit of 1 ppb (80).

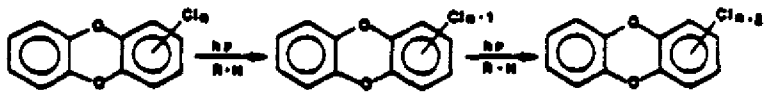
PHOTOLYSIS

There are basically three conditions necessary for the photoreduction of chlorinated aromatic compounds (81, 82):

- Presence of a hydrogen donor, usually in the solvent.
- Ultraviolet (UV) light of the appropriate wavelength.
- Absorption of the light.

In sunlight, only those compounds absorbing UV light above 290 nm can be expected to be subject to photodegradation. This includes the PCDDs and PCDFs.

TCDD photolysis proceeds via stepwise photoreductive dechlorination as follows (46, 81):



(rxn 10)

A hydrogen donor is essential; as a result, degradation is fast only in organic media, slow in water, and practically nonexistent on dry, inorganic surfaces (46). Most of the photolysis research has been conducted on organic solvents, such as methanol, hexane, diesel oil, or phenoxyether (46, 81, 83). In these solvents, the lower chlorinated isomers are extremely unstable to light. For instance, Crosby, et al. (46), have shown that 2,7-dichloro-, 2,3,7-trichloro-, and 2,3,7,8-tetrachlorodibenzodioxin in methanol will totally degrade within a few hours. The octachloro isomer was much more stable.

Shortly after the Seveso incident, it was demonstrated that olive oil is a suitable hydrogen donor (84). Total reductions in TCDD levels in excess of 60 percent were observed 48 hours after treatment with 400 l of olive oil emulsion per ha of contaminated soil. Furthermore, less than 0.07 percent of the applied olive oil was found in the leachate, indicating that TCDD could not be transported to lower geologic strata, even if it were totally dissolved in the oil film on vegetation or the ground.

Botre, et al. (85), evaluated various solvents to solubilize pure 2,3,7,8-TCDD and samples of contaminated soil from Seveso in aqueous solutions containing cationic, anionic, and nonionic surfactants prior to photolysis. The cationic surfactant, 1-hexadecylpyridinium chloride (HDPC), was found to be effective in solubilizing TCDD and in enhancing photolysis. The total destruction of 8 ug/ml 2,3,7,8-TCDD in 0.02M HDPC was accomplished in 4 hours.

Stanford Research Institute reported a half-life for gas-phase TCDD in sunlight of 5 to 24 days (55). When samples of TCDD were placed on dry soil and glass plate and irradiated with UV, no degradation was observed after 96 hours and 14 days, respectively (83). TCDD in the crystalline state resists photolysis (81). Thus, the presence of the hydrogen donor is essential to rapid photolysis.

In general, the rate of dioxin photolysis increases as the number of substituent chlorine atoms decreases (83). According to Plimmer, et al. (83), even the dioxin nucleus will photodegrade in methanol. On the other hand, Crosby (81) reports that monochlorodibenzodioxin is essentially stable to photolysis.

In 1981, 4300 gallons of wastes containing approximately 343 ppm each of TCDD (7.5 kg), trichlorophenols (50 to 55 percent), and ethylene glycol derivatives (45 to 50 percent) were subjected to a commercial-scale batch treatment UV-degradation process

for TCDD destruction. This destruction process involved the two following major steps: (1) multiphase extraction/neutralization with sulfuric acid/n-hexane/caustic soda (up to eight multiple extractions of the hexane, acid, and oily phases); and (2) photolytic reduction using UV-irradiation and isopropyl alcohol as a proton donor. The process was found to be effective in reducing the initial TCDD from 7.5 kg to less than 100 g (destruction efficiency of 98.24 percent).

The picture with regard to dibenzofurans is less clear. Crosby, et al. (46), reported that PCDFs follow the same degradation pattern as PCDDs, with higher chlorinated isomers being more stable. Hutzinger (86) reported that both 2,8-dichloro- and octachlorodibenzofuran in methanol and hexane photodechlorinated rapidly. Buser (45) stated that higher chlorinated PCDF isomers photodegrade faster.

INCINERATION

There has been little research conducted on thermal destruction of PCDD, and even less on PCDF. Different researchers have reported 2,3,7,8-TCDD destruction at 800°C (40, 55). In general, however, incinerator operating conditions currently considered adequate for TCDD destruction are a temperature of at least 1000°C and a dwell time of 2 seconds (33). At 700°C and a dwell time of 21 seconds, only 50 percent destruction was achieved; at 800°C and a dwell-time of 21 seconds, 99.5 percent destruction was achieved.

In 1972, tests were conducted to evaluate the feasibility of Agent Orange incineration (78). The incineration test unit consisted of a SUE® burner/incinerator and reaction tailpipe, a Venturi scrubber, a scrubber collection bank, and a gas sampling system. Both exhaust gas and scrubber liquid effluents were sampled and analyzed for TCDD. The test results showed that TCDD was not present either in the combustion gases or in the scrubber liquid at the detection limit of 15 ppb (78).

Recently, a truck-mounted mobile waste processor, developed by Pyro-Magnetics Corporation, was evaluated at the University of Tennessee Space Institute in Tullahoma, Tennessee (87). The mobile incinerator was fed a 41.7 percent PCB waste at the rate of 222 l/hour in four tests totalling 14 hours in duration. Average destruction and combustion efficiencies of PCBs were 99.9999 and 99.9979 percent, respectively. The incinerator outlet temperature was reported to average 1245°C, and the residence time was 2.9 seconds. The 2,3,7,8-TCDD was detected in only one of three test burns, and averaged 14 percent of the total TCDD; the 2,3,7,8-TCDF isomer averaged 27 percent of the total TCDF.

Ackerman and coworkers (30) have developed guidelines for land-based incineration of PCBs and related materials; the criteria are summarized in Table 4-1. For non-liquid PCBs or materials contaminated with PCDFs and/or PCDDs, there is an additional requirement: the mass air emissions must contain $\leq$0.001 g PCB/kg PCB fed to the incinerator

Table 4-1

COMBUSTION CRITERIA FOR PCBs AND PCDF/PCDD-CONTAMINATED WASTES

1. Maintenance of introduced liquids for 2-sec dwell time at 1200°C (±100°C) and 3 percent excess oxygen in the stack gas, or for 1.5-sec dwell time at 1600°C (±100°C) and 2 percent excess oxygen in the stack gas.
2. Combustion efficiency $\geq$99.9 percent based on $100[CO_2]/([CO_2] + [CO])$.
3. Rate and quantity of PCBs fed to combustion chamber must be measured and recorded at regular intervals (<15 min).
4. Incineration temperatures continuously measured and recorded.
5. Monitoring of stack emissions products for:
   a. O_2
   b. CO
   c. CO_2
   d. NO_x
   e. R-Cl (total chlorinated organic content)
   f. PCBs
   g. PCDFs and PCDDs
   h. Total particulate matter.
6. Automatic shutoff of waste flow when:
   a. Combustion temperature decreases to below conditions stated in Item 1 above.
   b. Failure of monitoring operations stated in Item 5 above.
7. Caustic scrubbers used for HCl control (or an approved alternate method).

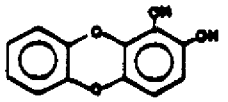
To date, M/T Vulcanus is the only incineration vessel that has been employed to incinerate TCDD-contaminated materials (78). The M/T Vulcanus is a double-hull, double-bottom tanker specifically designed to incinerate chlorinated hydrocarbon waste

products. Waste to be processed must be liquid in order to be pumped, although solid substances up to 2 in (5 cm) in size may be included. Two incinerators are installed aboard, each having an overall height of 34.25 ft (10.45 m) with an inner dimension of 15.75 ft (4.8 m) for the combustion chamber. The waste feed rate is about 12.25 tons/hour (12.5 tonnes/hour), and the residence time is approximately 1.0 second at 1500°C (78).

BIOLOGICAL DEGRADATION

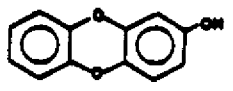
Investigations have shown that dioxins exhibit a relatively strong resistance to biodegradation (36, 50). Most of the work on dioxins has been focused on 2,3,7,8-TCDD because of its extreme toxicity. Approximately 100 strains of microbes, which had previously shown the ability to degrade persistent pesticides, were tested. Only five strains were shown to have the ability to degrade 2,3,7,8-TCDD (36, 188). It is possible that the lesser chlorinated PCDDs are more susceptible to biodegradation, as is the case with PCBs (50). TCDD was found to be practically immobile in soil, and adsorbed strongly on lake sediments (89, 90, 91, 92, 93, 94). Although 2,3,7,8-TCDD may be genuinely metabolized in aquatic ecosystems, the rate is extremely slow (62).

Kiecka and Gibson (1979) have reported that unsubstituted dibenzo-p-dioxin can be readily metabolized by a mutant strain of Pseudomonas (sp. N.C.I.B. 9816 strain II) when an alternative source of carbon, such as salicylate, is available (36). The dioxin molecule was metabolized first to a cis-1,2-dihydroxy-1,2-dihydrodibenzo-[1,4]dioxin



(rxn 11)

which was subsequently dehydrated to yield 2-hydroxydibenzo[1,4]-dioxin



(rxn 12)

as the major metabolite. They also reported finding no organisms capable of utilizing dibenzo-p-dioxin as the sole source of carbon.

Cannon and coworkers (95) evaluated microbial degradation of TCDD in soil samples from "Zone A" at Seveso. Soil samples containing an average of 0.1 ppm TCDD were analyzed in the presence and absence of organic compost. The results indicated a 25 percent reduction in TCDD concentration in 480 days. Studies by Kearney, et al. (96), have indicated that the average TCDD remaining in the soil after 1 year of weathering was approximately 50 percent at all concentrations tested (1 to 10 ppm). However, it has been determined that microbial degradation is not a major factor in the environmental degradability of these toxins. The predominant mode of elimination of this compound within the environment appears to be in the photodecomposition by sunlight (88).

On the other hand, research by Hutter (97) and Young (98) indicates that microbial degradation of TCDD is very slow under the most optimum conditions, leading to the conclusion that dioxins are essentially nonbiodegradable. Furthermore, these researchers suggest that the half-lives for dioxins in soil observed in earlier experiments are actually the result of drastically decreased extractability, and do not reflect a loss of material from the soil.

Recent research has shown that dioxins are metabolizable by certain mammals (99). The biotransformation of TCDD in dogs appears to be a detoxification process. Experiments with guinea pigs showed that the acute toxicity of the metabolites was at least 100 times lower than that of the parent compound. Conversely, when predioxins were administered to animals, there was no indication of metabolism-induced dioxin formation (C. Rappe, Personal Communication).

Little research has been conducted on dibenzofuran biodegradation. However, the metabolism of chlorinated biphenyls may produce penta- and hexa-CDFs (64). The researchers have suggested that the PCDFs were synthesized via ortho-hydroxylation in association with chlorine migration, and have further indicated that it may have a substitution pattern at positions 1, 2, 3, 7, 8, and 9. This newly discovered phenomenon is of great toxicological significance. There is also evidence to suggest that tetra-CDFs may be formed by liver metabolism in rats. These CDFs are potentially much more toxic than the parent chlorobiphenyls, even if their production is via a minor metabolic pathway. The metabolism of PCBs has often been regarded as a detoxification process. This is particularly so when the metabolites are conjugated and excreted.

Section 5

ENVIRONMENTAL PERSISTENCE

With their high toxicities, the environmental fates of PCDDs and PCDFs are of increasing importance. Unfortunately, little is known of the environmental behavior of most of these compounds. Only 2,3,7,8-TCDD has been studied in any detail, and then often with inconclusive results (100). To what degree the findings with TCDD are applicable to other PCDDs and PCDFs is largely undetermined.

Primarily as a result of the Seveso incident, most of the environmental research has centered on the persistence of TCDD in soil. In general, TCDD is highly persistent in soils (32). According to Di Domenico, et al. (101), there is a high initial loss rate due to volatilization and photodecomposition of TCDD present on the surface, followed by a very slow steady-state loss rate. They estimate that after 10 years, 30 to 44 percent of the TCDD present after the first half year would still be in the soil. Since other researchers have indicated that up to 50 percent can be lost in the first year, this emphasizes the extremely slow loss rate in subsequent years (55, 96).

Once the TCDD has been washed into the soil, it is protected from the influence of solar photolysis and volatilization (102, 103). It is not easily washed out of soils, being virtually immobile in all but very sandy soils (7). The role of such processes as erosion as a transport mechanism is unknown (50). TCDD is not taken up by plants to any significant degree (7, 55), and there is relatively little biomagnification in mammals (50). As noted in the previous section, these compounds are not readily biodegradable. Stehl, et al. (104), have suggested that 2,3,7,8-TCDD is probably stable toward air oxidation.

There appears to be only one significant environmental decomposition mechanism: photolysis. As noted earlier, however, a hydrogen donor is necessary for this reaction. Consequently, TCDD deposited on dry surfaces may be resistant even to photolysis. Crosby (81) demonstrated at Seveso that spraying a contaminated environment with an innocuous organic solvent (in this case, olive oil) can greatly enhance TCDD decomposition.

If TCDD is washed into a water body, it will strongly adsorb onto bottom sediments (55). There is some evidence to suggest that PCDDs and PCDFs will bind stronoly to any particle. No data is available on the solubilities of the sorbed PCDDs and PCDFs. Neither PCDDs nor PCDFs are particularly soluble in water, and solubility decreases with increasino chlorine saturation (64). How the solubility of PCDDs is affected by the presence of colloids or solvating agents in natural waters is unknown (50). However, there is some evidence that TCDD bioaccumulates, suggesting at least a limited mobility through the food chain (7, 100). Fish do not seem to biomagnify TCDD to any great extent through food consumption (50). However, there is evidence to indicate that bioaccumulation from the water vector can exceed 6000 times the water content (105). Nevertheless, different isomers benave differently, and the extent and impact of these differences require further investigation (50). Table 5-1 provides evidence of the lower solubility of the higher chlorinated isomers. In addition, Table 5-1 shows the benavior of different isomers in fish.

Several researchers have speculated on various environmental properties of TCDD, based on the properties and fate of other similar organic compounds (see Table 5-2). These have then been used to predict persistence and fate in a model aquatic ecosystem. Where testable, these predictions have been reasonably accurate. However, in the absence of more extensive test data, these constants remain speculative.

Table 5-1

PCDD AND PCDF CONCENTRATION RANGE IN HUDSON AND HOUSATONIC RIVER SEDIMENTS AND FISH FROM WOODS POND (106)

PCDDs	River Sediments (ng/g)	Woods Pond Sediments ng/g	Woods Pond Yellow Perch ng/g
Cl_4	ND* - 44		
Cl_5	ND - 235		
Cl_6	ND - 1,335		
Cl_7	ND - 18,950		
Cl_8	3 - 15,550		
PCDFs			
Cl_4	ND - 200	<0.005	1.36
Cl_5	ND - 193	0.309	0.58
Cl_6	ND - 377	0.15	0.46
Cl_7	ND - 2,436	0.92	<0.005
Cl_8	ND - 1,310	0.27	<0.005

* ND = None Detected.

Table 5-2

SUMMARY OF PREDICTED PARTITION, TRANSFER, AND DEGRADATION CONSTANTS FOR 2,3,7,8-TCDD (50)

Volatilization (25°C) ($cm^{-1}_{-d}{}^{-1}$)	1.7×10^2
Photolysis (45°N, June) ($cm^{-1}_{-d}{}^{-1}$)	
Attenuation: High	0
Low	0
Sorption	
Partition (100% organic)	1.6×10^6
(10% organic)	1.6×10^5
Transfer (g water (g sediment)$^{-1}_{-d}{}^{-1}$)	
(100% organic)	1.0×10^2
(10% organic)	1.0×10^1
Bioaccumulation	
Partition	3.3×10^4
Transfer (d^{-1} $g^{0.2}$)	
Uptake	1.9×10^2
Clearance	5.9×10^{-3}
Fraction Degraded	0

MONS 215246

Section 6

PCDD/PCDF/PCBP TOXICOLOGY

INTRODUCTION

The following section discusses the toxicological effects of PCDDs, PCDFs, and PCBPs on laboratory animals and man. On a molecular basis, 2,3,7,8-TCDD is perhaps the most toxic synthetic molecule known to man (33). Only bacterial exotoxins (i.e., Botulinum Toxin A, tetanus toxin, and diphtheria toxin) are more potent poisons (107).

Both higher and lower chlorination reduce toxicity, but the toxic symptoms of the various isomers are similar, the only difference being the amount required to produce a given effect.

The isomers with the highest acute toxicity are 2,3,7,8-tetra-CDD, 1,2,3,7,8-penta-CDD, 1,2,3,4,7,8-, 1,2,3,6,7,8-, and 1,2,3,7,8,9-hexa-CDD, 2,3,7,8-tetra-CDF, 1,2,3,7,8- and 2,3,4,7,8-penta-CDF, and 1,2,3,4,7,8- and 2,3,4,6,7,8-hexa-CDF (see Figure 6-1). This figure clearly illustrates the current thought that full lateral substitution with Cl in the 2,3,7,8-positions leads to high toxic potency. Most of the research has centered on the 2,3,7,8- isomers.

ANIMAL TOXICITY

Rodents

Comparative oral lethal dose values for various PCDDs in mice and guinea pigs clearly show that 2,3,7,8-TCDD and 1,2,3,7,8-penta-CDD isomers are the most toxic (108). The toxic effects induced by PCDDs vary both quantitatively and qualitatively among different species (33, 108, 109, 110). The major characteristics of lethal doses of 2,3,7,8-TCDD in all animal species are progressive weight loss and general weakening, with death occurring within a few weeks or months (111). Degeneration of the liver is also a common pathologic effect (112). Hepatic necrosis produced by 2,3,7,8-TCDD is probably a contributing cause of death in rats and rabbits. However, the number of deaths attributable to liver insufficiency is lower in mice, and minimal in guinea pigs (108).

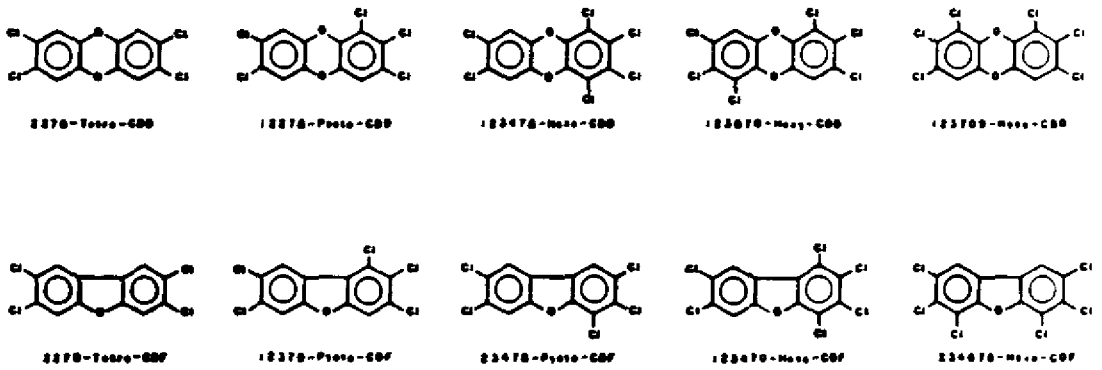


Figure 6-1. The Most Toxic PCDD and PCDF Isomers

There is minimal damage to the liver of guinea pigs, the species most sensitive to 2,3,7,8-TCDD toxicity. This suggests that 2,3,7,8-TCDD is probably affecting some fundamental process in animal cells, which leads to a general dysfunction of those cells (111).

A large percentage of 2,3,7,8-TCDD persists in the liver in unmetabolized form, particularly concentrated in the microsomes and microsomal fractions (fragments of ribosomes and endoplasmic reticulum) (33, 109, 118). These findings suggest that the unmetabolized 2,3,7,8-TCDD, rather than a metabolite, is responsible for the observed toxic effects (115).

Various experiments conducted to date have failed to provide the exact biochemical mechanism(s) of 2,3,7,8-TCDD lethality. Adverse effects on the following processes do not appear to be the mechanisms by which 2,3,7,8-TCDD exerts its toxicity (33, 111):

- DNA synthesis.
- Protein synthesis.
- ATP production.
- Oxidation-reduction state of cells.
- Action of thyroid and glucocorticoid hormones.
- General absorption of carbohydrates, amino acids, and fatty acids from the intestine and the transfer through cell membranes.
- Aerobic and anaerobic metabolism of carbohydrates, amino acids, and fatty acids.
- Fatty acid composition of tissues.

At this time, the most reasonable hypothesis is that 2,3,7,8-TCDD binds to a receptor in the cytosol (soluble portion of the cytoplasm) fraction of cells, is transferred into the nucleus, and increases or decreases the synthesis of a critical protein(s) or enzyme(s). This increase or decrease in crucial protein or enzyme levels eventually leads to a general dysfunction of a number of different cell types, resulting in death (111). However, the receptor theory may not explain the chloracnegenicity of TCDD. Receptor proteins have not been identified on skin cells.

The lower toxicity of 2,3,7,8-TCDD in rats, as compared to other mammals, suggests some degree of metabolic transformation of the parent compound (33, 115, 116, 117).

The concentration of dioxin in the liver of rats may be five times greater than that found in their body fat. Concentrations within the kidney, thymus, and spleen are 1/12 to 1/50 of that found in the liver (33). The substance which is not retained is excreted unchanged with the feces (60 percent) via the bile fluid. A small amount (5 percent) is excreted in the urine as a water-soluble conjugate (109). This suggests that 2,3,7,8-TCDD metabolism may be occurring in the fat, since water-insoluble compounds, like dioxins, are excreted predominantly, if not exclusively, through the feces (33, 115).

Female albino rats (Porton strain), given a single oral dose of 200 ug/kg 2,3,7,8-TCDD (their approximate LD-50), died within 3 to 9 weeks following a period of weight loss and depressed food intake (116). Within 24 hours after the dose was administered, there were changes in the hepatic parenchymal cells, including a proliferation of the smooth endoplasmic reticulum, and alterations in the metabolism of various substrates. Approximately 4 weeks after the 2,3,7,8-TCDD was administered, giant multinucleated cells were found within the area of the centrilobular zone (116). These cells were probably formed by the fusion of preexisting degenerate parenchymal cells (109, 117). This hypothesis is supported by the following evidence:

- No mitoses occurred within these cells.
- These cells tend to follow the line of trabeculae.
- Isolated bile canaliculi were occasionally observed to be surrounded by the cytoplasm from the multinucleated cells.
- Adjacent mononucleated parenchymal cells showed areas of apparent fusion of their plasma membranes.

In addition, there were increased numbers of lysosomes, loss of microvilli from the bile canaliculi, and proliferation of the smooth endoplasmic reticulum in the centrilobular parenchymal cells (116). These lesions disappeared in surviving animals over a period of time.

Histochemically, there was a centrilobular loss of ATPase activity within 3 days. This loss extended to the periportal area by the sixth week (116). Biochemical studies on plasma membranes have indicated that the Na^{+}/K^{+} ATPase is unaffected, whereas the K^{+}/Mg^{+2} ATPase activity is drastically reduced (109, 116).

It is known that the K^{+}/Mg^{+2} ATPase of the sarcoplasmic reticulum requires a lipid for activation; the degree of activation can be affected by the type of lipid involved (116). Toxic levels of PCDD result in a chronic increase in triglycerides and development of a fatty liver (108). It is possible that the initial disturbance of the oxidative enzymes of the endoplasmic reticulum leads to alterations in endogenous lipid metabolism, with a consequent change in the structure and function of the parenchymal cell membrane. These changes could lead to hepatic insufficiency with toxic effects observed macroscopically.

2,3,7,8-TCDD and other halogenated PCDDs stimulate a number of enzyme activities, most notably within the liver (108, 111, 114, 118, 119). Hepatic δ-aminolevulinic acid (ALA)* synthetase and aryl hydrocarbon hydroxylase (AHH)** are induced by the presence of PCDDs, particularly 2,3,7,8-TCDD (108, 120). The PCDDs which have been found to induce ALA synthetase appear to have two common properties: (1) halogen atoms occupy at least three of the four ring positions (i.e., 2, 3, 7, 8); and (2) at least one free nonhalogenated carbon atom is unoccupied (108, 118). Available toxicological data indicate that those PCDDs that are not toxic generally do not induce ALA synthetase; those which are lethal, even at extremely low concentrations, induce the enzyme. 2,3,7,8-TCDD has been found to be many orders of magnitude more potent than any other known inducer of ALA (33, 118).

Microsomal monooxygenase (e.g., AHH) is a membrane-bound enzyme complex which catalyzes the oxidative (and for certain substrates, reductive) metabolism of numerous xenobiotics and endogenous lipophilic compounds (118). 2,3,7,8-TCDD is a highly lipophilic macromolecule (120). The induction of AHH by various PCDDs is carried out by the same mechanism as that found for the induction of ALA (118). The hypothesis that the induction of ALA and AHH by 2,3,7,8-TCDD is mediated by the interaction of the ligand with an induction receptor (the product of a regulatory gene that combines with a small molecule to initiate gene expression) is supported by the following evidence (118):

- The potency of 2,3,7,8-TCDD in evoking a dose-related induction of ALA and AHH activity.
- Chemical specificity.

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* The initial and rate-limiting enzyme in heme synthesis.

** A cytochrome P_1-450 mediated monooxygenase.

- Biological specificity (i.e., a specific type of microsomal monooxygenase activity is induced in some, but not all, tissues).

If the hepatic cytosol-binding species is in fact the induction receptor, a possible mechanism by which the receptor ligand complex in the cytosol initiates an increase in nuclear activity would involve the translocation of this complex into the nucleus in a manner analogous to the mode of action ascribed to steroid hormone receptors (33, 118).

Theoretically, the most logical pathway of 2,3,7,8-TCDD metabolism is by hydroxylation at the 1, 4, 6, or 9 position of the molecule by AHH. The hydroxylation of aromatic rings in positions adjacent to halogen atoms is an uncommon reaction of the cytochrome P-448 monooxygenase system. This most likely accounts for the metabolic stability of 2,3,7,8-TCDD within mammalian systems (115, 116). However, it has been postulated that 2,3,7,8-TCDD moves into a cell and binds to a specific cytosolic receptor. The receptor-dioxin complex then moves into the nucleus of a cell, where it initiates the synthesis of a specific messenger RNA, which directs the synthesis of cytochrome P-448. Other 2,3,7,8-TCDD molecules can then react with newly formed cytochromes, possibly to produce reactive intermediates that reduce or impair cellular metabolism, gradually inducing cell death (33, 120). These metabolites may be excreted as innocuous by-products which may affect specific cells in other organs, or may act as carcinogens or cocarcinogens (33).

If the activity of AHH were increased, more 2,3,7,8-TCDD would be metabolized. If 2,3,7,8-TCDD, rather than a metabolite, is responsible for the toxicity, then increased metabolism by the P-450 monooxygenase system should lead to a decrease in toxicity. Conversely, if a metabolite(s) is responsible for the toxicity of 2,3,7,8-TCDD, then an increase in metabolism may result in an increase in toxicity (115, 116). Several studies have shown that 2,3,7,8-TCDD induces many enzyme systems while suppressing others. 2,3,7,8-TCDD induces the following enzymes in addition to those previously mentioned (33, 108, 110):

- UDP glucuronyl transferase.
- Aldehyde dehydrogenase.
- Glutathione transferase B.
- DT-diaphorase.
- Benzopyrene hydrolase.

- Glutathione S-transferase.
- Ethoxycoumarin deethylase.

2,3,7,8-TCDD reduces the activity of the following enzymes (89):

- UDP-glucuronic acid pyrophosohatase.
- D-glucuronolactone dehydrogenase.
- L-gluconate dehydrogenase.

A sublethal dose of 2,3,7,8-TCDD in rats will cause a temporary increase in triglyceride and free fatty acid levels, with a persistent decrease of sterol esters. Lethal concentrations result in a fatty liver, increased serum cholesterol esters, and free fatty acids, with little change in triglyceride levels. These changes are due in part to the damage sustained by lysosomes (33). The decrease in the acid lipase activity, derived from damaged lysosomes, probably accounts for the increased levels of cholesterol esters within the liver. Therefore, another mechanism by which 2,3,7,8-TCDD may exert its toxic effect is by the increase of lipofuscin pigments (33). Lipid peroxidation, which precedes the formation of polymeric lipofuscins, is known to seriously damage membranous subcellular organelles, including lysosomes (33, 108, 121).

In one or more species of laboratory animals, bone marrow hypoplasia, testicular degeneration, renal pelvis and urinary bladder hyperplasia, and hemorrhage in the intestines and adrenals have been observed (108). Chronic administration of low levels of 2,3,7,8-TCDD to rats increeses the incidence of neoplasia (tumors) (108). The high incidence of tumors in rats fed subacute levels of 2,3,7,8-TCDD (i.e., 1 ppt to 5 ppb)* suggests the carcinogenic potential of the compound (112). Lung tumors that had been observed were characterized by squamous metaplasia of the bronchial epithelium and keratinization. Lymphocytic leukemia wes associated with a massive enlargement of the spleen. The most malignant tumors were observed in the lungs, kidneys, liver, and the skeletal musculature. Other neoplasms were found in the brain, skin, testes, and ears (33, 108, 112). The only abnormalities of the hematopoietic system noted in rats have been hemoconcentration and thrombocytopenia

* 1 part per trillion = 10^{-12} g TCDD/g food.
  1 part per billion = 10^{-9} g TCDD/g food.

(increase in the relative volume of red blood cells per unit volume of circulating blood and decreased number of platelets per unit volume of blood) (33).

The terms "embryotoxicity" and "fetotoxicity" denote all transient or permanent toxic effects induced in an embryo or fetus, regardless of the mechanism involved (33). A special fetotoxic effect is teratogenicity, which may be defined as an abnormality (likely irreversible) caused by impairment of an activity which is typical of embryonic or fetal development (33, 122). This effect may either be obvious by macroscopic appearance or "hidden" (micromorphological defect or mental abnormality). 2,3,7,8-TCDD is a specific teratogen, interfering only with a few special developmental processes (122).

Recent studies have shown that 2,3,7,8-TCDD can have a dramatic effect upon the hormones involved in reproduction (33). As little as 0.3 ug/kg of 2,3,7,8-TCDD injected into mice was found to increase the frequency of cleft palate (a teratogenic effect) (33, 122). This is by far the smallest effective dose of any teratogen known (122). Oral administration of 2,4,5-T containing dioxin to golden hamsters on the sixth to tenth day of gestation resulted in an increased absence of eyelids (33). Other fetotoxic effects of 2,3,7,8-TCDD include thymic atrophy, fatty infiltration of the liver, general edema, delayed head ossification, low birth weight, fetal reabsorption, and kidney abnormalities (33). Fetal mortality, early and late fetal reabsorptions, and fetal intestinal hemorrhaging occurred in rats given a 2,3,7,8-TCDD dosage as low as 0.125 ug/kg (33). The fetal kidney anomaly can be best described as a renal papilla which is markedly reduced in size, resulting in an enlarged renal pelvis (108). This may progress into hydronephrosis during metanephric kidney formation (123). The teratogenic effect of 2,3,7,8-TCDD may be a result of mutagenesis by intercalation of the parent compound into DNA, or by intercalation and covalent binding of the metabolite, analogous to findings of tests conducted with acridine and aminofluorene compounds (120).

Information implicating 2,3,7,8-TCDD as a mutagen is both scarce and conflicting (33). Derivatives of dioxin have shown negative results when tested for dominant lethal effects in rats, and only slightly positive results when tested for their capacity to produce chromosomal aberrations in bone marrow cells of rats (33, 108).

Studies on mice indicate that sublethal doses of 2,3,7,8-TCDD increase the number of erythrocytes (and their cell volume) and neutrophils, and decrease hemoglobin, serum

protein, and globulin levels. The number of blood platelets decreases, resulting in diminished clot reaction (109).

Another effect of 2,3,7,8-TCDD is its interaction with iron metabolism (33). Rats given 1.7 ug of the compound intragastrically have shown a twofold increase in the serosal transfer of iron, whereas no change was observed in mucosal iron uptake. However, it appears that this iron deficiency protects mice from many of the toxic effects exhibited by 2,3,7,8-TCDD. Mice rendered iron-deficient were found to be protected from elevated porphyrin levels and liver damage. Since mixed-function oxidative enzymes were elevated in iron-deficient mice, it was speculated that the depleted storage of iron within the tissues was responsible for the observed reduction in toxicity (33).

2,3,7,8-TCDD, 2,3,7,8-TCDF, and 2,3,6,7-TCBP are the approximate isostereomer of 3,4,3',4'-TCB. It is thus believed that the structural configurations that render TCDDs, TCDFs, and TCBPs toxic are analogous to those for TCBs. Namely, 3,4,3',4'-TCB may assume a planar or nearly planar configuration with its four chlorine atoms projecting from the lateral ring positions, similar to 2,3,7,8-TCDD. However, if chlorine is substituted in a position adjacent to the biphenyl bridge (e.g., 2,4,3',4'-TCB), this sterically hinders the biphenyl rings and prevents planarity. Such nonplanar PCB analogs are incapable of initiating AHH activity (124). Thus, among the halogenated biphenyls, there are two structural requirements for the induction of AHH: (1) the presence of at least two adjacent halogen atoms in the lateral position of each benzene ring (e.g., positions 3,4,3',4' or 3,4,5,3',4',5'), and (2) the absence of halogen atoms adjacent to the biphenyl bridge (positions 2, 6, 2', 6'). The effects of different biphenyls on AHH are shown in Appendix B.

Birds

It has been shown that there is a very good correlation between the potency of PCDDs and PCDFs to induce AHH activity and their capacity to produce chloracne, thymic atrophy, and edema in chickens (124).

Histopathological examinations of birds exposed to 2,3,7,8-TCDD have revealed atrophy of the spleen, involution of the thymus, decreased numbers of lymphocytes (particularly in the bursa of Fabricius), pulmonary edema (i.e., myocardium), and necrosis of the liver (50). The disease known as "chick edema" has also been reported in TCDD-exposed birds. This disease is characterized by the accumulation of serous fluids in the ascites, anascara, and pericardial sac (125, 126). One study showed

that 3-day-old chicks treated with doses of 1 and 10 ug of 2,3,7,8-TCDD/kg body weight developed chick edema (125); the chicks also exhibited dyspnea, accumulation of mucus in the mouth, and enlargement and mottling of the liver (126). Alterations in drug-metabolizing enzymes such as the cytochrome P-450 system and the accumulation of porphyrin were also noted.

The biological potency (ED_{50}) of 2,3,7,8-TCDD and various biphenylene and biphenyl congeners was estimated from a log dose-response curve for the induction of hepatic aryl hydrocarbon hydroxylase activity in a chicken embryo (124). ED_{50} values for the three halogenated biphenyls found to induce AHH (i.e., 3,4,3',4'-TC8; 3,4,5,3',5'-HC8; and 3,4,5,3',4',5'-H88) were reported to average 4.5 nmoles per egg. In comparing the potency of 3,4,3',4'-TCB with that of 2,3,7,8-TCDD and Aroclor 1254, 2,3,7,8-TCDD was almost 500 times as potent as 3,4,3',4'-TC8, and 2 x 10^5 times as potent as the commercial PCB mixture (124).

2,3,6,7-TC8P was found to be a very potent inducer of AHH activity (ED_{50} = 14 pmoles per egg), as was 2,3,6,7-tetrabrombiphenylene (TB8P) (ED_{50} = 22 pmoles per egg). Both of these biphenylenes were reported to be approximately equipotent to 2,3,7,8-TCDD and TCDF. OCBP, 2,3,6,7-tetramethoxybiphenylene, and 2,7-dinitrobiphenylene (DNBP) could not induce AHH activity at doses of 46 nmoles per egg (124). 2,3,6,7-TCBP was also found to be roughly equipotent to 2,3,7,8-TCDD in inducing AHH activity in both mouse and rat livers.

The ability of biphenyls and biphenylene congeners to compete for sterospecific binding of [^{3}H]TCDD to mouse and rat liver cytosol was also investigated (124). 2,3,6,7-TC8P and 2,3,6,7-TB8P bound avidly, whereas 3,4,3',4'-TCBP proved to be approximately 30 times less potent.

Table 6-1 compares the binding affinity in vitro, K_D (for mouse liver cytosol), and the biological potency in vivo, ED_{50} (for hepatic AHH activity in a chicken embryo), for 2,3,7,8-TCDD, several biphenylenes, and one biphenyl congener. As shown, 2,3,6,7-TCBP and 2,3,6,7-TB8P are roughly equipotent to 2,3,7,8-TCDD. The other biphenylene analogs failed to compete with [^{3}H]TCDD for hepatic cytosol binding sites or for inducing AHH activity (124). It should be noted that all of the biphenylene and biphenyl congeners which failed to induce AHH activity also did not exhibit any affinity for the cytosol binding sites. In contrast, the three halogenated biphenylenes and biphenyls which were found to induce AHH activity also competed with [^{3}H]TCDD for specific cytosol binding sites (124).

Table 6-1

BIOLOGICAL POTENCY AND BINDING AFFINITY OF
VARIOUS BIPHENYLENE AND BIPHENYL CONGENERS (124)

Compound	Biological Potency in Vivo ED_{50} (nmol/kg)	Binding Affinity in Vitro (K_n [nM])
TCDD	0.20	0.27
2,3,6,7-tetrachlorobiphenylene	0.28	0.34
2,3,6,7-tetrabromobiphenylene	0.44	0.47
3,4,3',4'-tetrachlorobiphenyl	88	15
Octachlorobiphenylene	Inactive (470)*	Inactive (540)*
2,7-dinitrobiphenylene	Inactive (930)*	Inactive (540)*

* The highest concentration tested that was judged to be soluble.

Fish

A considerable amount of extrapolation is required in order to interpret the available data on fish, which pertains almost entirely to lethality (50). It appears that lethal levels of 2,3,7,8-TCDD in fish are within the same order of magnitude as those described for mammals (50). Rainbow trout that were fed 2.3 mg/kg 2,3,7,8-TCDD (representing a total dose of approximately 700 ug/kg body weight) exhibited a 50 percent mortality rate within a 60-day period. Exposure of rainbow trout to 0.1 ug/l of water over a 96-hour treatment period (representing a total dose of approximately 600 ug/kg body weight) resulted in 50 percent mortality within a period of 21 days (50). In comparative experiments with Coho salmon, a 96-hour exposure to an initial water concentration of 0.105 ug/l (representing an estimated total dose of 5.4 ug/kg body weight) was found to be lethal within a period of 20 days (50).

There was no significant increase in egg mortality in either rainbow trout or pike when exposed to water levels as high as 0.01 ug/l over a 96-hour period (50). However, there was a significant increase in the mortality rate of yolksac-fry in both of these species.

An 80 percent mortality rate was observed in the feeding-fry of orke at an exposure of 0.01 ug/l of water over a 96-hour treatment period (50). There was no significant difference in the same exposure regime for rainbow trout. Reported histopathological responses include edema, rupturing of blood vessels, and necrotic livers. Teratogenic changes in embryos include shortened maxilla and structural defects of the operculum (50).

Monkeys

The most striking characteristic of animals exposed to dioxin is the substantial loss of body weight (109, 127). In addition to progressive weight loss, monkeys exhibit puffiness of the eyelids (110, 127, 128). Dryness and granularity of the skin, loss of hair, and reduced physical activity have also been observed (128). Two types of lesions have been reported in all laboratory animals studied: (1) involution of the thymus, and (2) testicular alterations, including atrophy, necrosis, and abnormal spermatocyte development (33). It is believed that 2,3,7,8-TCDD has a suppressive effect on testicular microsomal AHH. Two other hyperplastic lesions in monkeys appear to be related to 2,3,7,8-TCDD or its metabolites: (1) bile duct hyperplasia, and (2) epithelial hyperplasia of the renal pelvis, urinary bladder, and ureter (110). Only one lesion, hypertrophic gastritis, has been observed in primates (33). This lesion is characterized by chronic inflammation of the gastric mucosa, which occurs in the fundic and pyloric regions, combined with small gastric ulcers penetrating the mucosa (33).

In experiments with rhesus monkeys exposed to dioxins, researchers found reduced hematopoiesis, degeneration of blood vessels, focal necrosis of the liver, gastric ulcers, and reduction of spermatogenesis. Under gross observation, experimental monkeys exhibited obvious dilation of the heart, especially on the right side. Under microscopic examination, fibers of the cardiac muscle were distinctly separated by fluid, and individual muscle cells were hypertrophic, with enlarged, distorted, and hyperchromic nuclei. Although the lungs were not appreciably altered, isolated areas of atelectasis, congestion, edema, and fibrosis were observed. Their livers were small, firm, and moderately yellow, with many enlarged, multinucleated parenchymal cells. Necrosis of parenchymal cells occurred in the centrilobular zone, and some areas of fibrosis were apparent in the periportal area. Their spleens were found to be small; the germinal centers were surrounded by only scattered lymphocytes, and the blood sinuses were practically devoid of cells. The seminiferous tubules of the testes had abundant spermatogonia and sertpli cells.

However, only a few primary spermatocytes were present, and no spermatids or mature spermatozoa were observed (33).

The mesenteric lymph nodes of the monkeys were light tan and edematous. Microscopically, the nodes resembled a splenic disarray of cellular architecture. Under gross observation, the bone marrow resembled coagulated plasma. Microscopically, only a few hematopoietic cells were seen within the marrow; these were equally divided between members of the myeloid and erythroid systems. Changes in the skeletal muscle resembled those of cardiac muscle. Facial edema and petechiae were commonly observed (33).

Whereas rats store 2,3,7,8-TCDD mainly in their livers (40 percent versus 10 percent in monkeys), monkeys accumulate the toxin in lipid tissues such as the skin, muscle, and fat (109). Consequently, unlike rats, the number of deaths attributable to liver insufficiency is minimal in monkeys (108). Rhesus monkeys that were fed 500 ppt 2,3,7,8-TCDD on a daily basis developed signs of toxicity at intake levels as low as 1 ug/kg body weight (112). A total intake of 3 ug/kg over a period of 9 months was sufficient to cause death (112, 127). The LD-50 of monkeys exposed to a single oral dose of 2,3,7,8-TCDD is approximately 50 to 70 ug/kg, with death occurring within 45 days (127). Prior to death, their peripheral blood is practically devoid of immature red and white blood cells (127). The marked thrombocytopenia was associated with widespread hemorrhaging (127). While death is usually associated with severe pancytopenia in chronically exposed animals, hematological changes do not always occur in those acutely exposed. Widespread hemorrhaging occurs only in chronically exposed animals (127).

It appears that damage is greater to monkeys and other experimental animals when submilligram doses of toxin are administered over a period of time, rather than all at one time (128). It has thus been speculated that toxicity is more closely related to the length of time that the compound is able to interact than to the amount of toxin administered (127, 128). As exposure time lengthens, the severity of cellular deterioration in bone marrow and lymphoid tissue becomes widespread (127). The observed ventricular dilation and myocardial hypertrophy may in part be related to the reduction in circulating erythrocytes and the subsequent increase in cardiac workload (127). Focal areas of hemorrhage were found in the epicardium, myocardium, and endocardium (127). Petechial hemorrhages were observed in the atria and the tips of the papillary muscles (127).

The possibility of reproductive abnormalities also exists. Altered levels of serum progesterone and estradiol, associated with both difficulties in conception and early abortions, have been reported in female monkeys exposed to low levels of 2,3,7,8-TCDD (127) Testicular atrophy may also develop in male monkeys given small amounts of dioxin (127).

Considerable edematous fluid usually surrounds the epithelium of those exposed to 2,3,7,8-TCDD. Inflammatory cells, with a predominance of neutrophils, have been observed to be in close proximity to ruptured submucosal mucinous cysts (127). The rupture of such cysts produces ulcers of the gastric mucosa (127). Metaplastic changes, characterized by numerous mucous-secreting cells, have been observed in the ductal epithelium of the salivary glands, bile ducts, and pancreatic ducts. Similar changes may also occur in the bronchial epithelium and the palpebral conjunctivae (127). Hemorrhages within the brain have been limited to the Verchow-Robbin's spaces surrounding the blood vessels (127).

A series of single oral exposures were performed on female rhesus monkeys to establish a toxic (acute) dose for TCDF. The LD-50 value was determined to be 1,000 ug/kg, a dose approximately 20-fold higher than that estimated for 2,3,7,8-TCDD (129). Toxicity was characterized by the onset of the following symptoms 7 to 10 days after exposure (129):

- Progressive weight loss.
- Facial edema.
- Loss of eyelashes and nails.
- Development of white streaks in the eyelids.
- Thickening of the skin.
- Death within 2 to 4 weeks.

The disease pattern cannot be distinguished from that produced by PCBs or 2,3,7,8-TCDDs (107, 128, 129).

Data pertaining to lethality, carcinogenicity, and reproductive effects are summarized in Table 6-2 (50).

Table 6-2

SUMMARY OF THE DOSAGE ASSOCIATED WITH THE TOXICOLOGICAL CRITERIA FOR VARIOUS PCDD CONGENERS

		Acute Lethality				AHH Induction (Rat)	Carcinogenicity (Rat) (ug/kg-bw d)	
		LD_{50} (ug/kg-bw)		Relative to 2,3,7,8-TCDD				
Homologue	Isomer	Vertebrates	Guinea Pig	Guinea Pig	Mouse	Relative to 2,3,7,8-TCDD	Minimum Effect Dose	No Observed Effect Dose
D_2CDD	2,7	>2,000,000				33,000		
	2,8	300,000	>300,000	>150,000		33,000		
T_3CDD	2,3,7	30,000	30,000	15,000	>1,500	3,100		
T_4CDD	2,3,7,8	2-5,000	2	1	150	1	1×10^{-2}	1×10^{-3}
	1,3,7,8					240		
	1,2,3,8					1,700		
	1,2,3,4	<1,000,000				33,000		
P_5CDD	1,2,3,7,8	3-338	1	1.5	170	15		
	1,2,4,7,8	1,100-5,000	1,100	560	>2,500	330,000		
H_6CDD	1,2,3,4,7,8	70-800	73	40	410	21	36×10^{-2}	
	1,2,3,6,7,8	70-1300	70-100	40	630	85		
	1,2,3,7,8,9	60-1400	60-100	40	>720	130		
	1,2,3,6,7,9					220,000		
H_7CDD	1,2,3,4,6,7,8	>400	>400	>300		370		
	1,2,3,4,6,7,9					10,000		
O_8CDD		>1,000,000				53,000		

HUMAN TOXICITY

Chloracne, an often disfiguring and persistent dermatological disorder, is caused by human exposure to several chlorinated aromatic compounds, including PCBs, PCDFs, and PCDDs (108, 130). This anomaly is analogous to hyperkeratosis in mice, rats, rabbits, and monkeys (108, 130). Chloracne may appear weeks, months, or even years after initial exposure to the toxin (33, 108). According to Moore (131), chloracne is the most sensitive indicator of poisoning in the human subject. However, the absence of chloracne does not preclude intoxication. Exposure to 2,3,7,8-TCDD may also cause polyneuropathy (nerve disease) and nystagmus (rapid eye movement).

Liver dysfunction is manifested by hepatomegaly and elevation of enzyme levels (33). Daily human exposure to 0.01 ug of 2,3,7,8-TCDD may result in incipient carcinogenicity, but existing evidence indicates that PCDDs are at most only mildly carcinogenic (107). Exposure to 4 ug of 2,3,7,8-TCDD would result in a shortened lifespan, and daily exposure to 290 ug would likely cause acute toxicity (33).

The results of laboratory studies on 2,3,7,8-TCDD and its interaction with iron metabolism in rats (described in the preceding subsection) have significant implication for toxicity in humans. Persons with a high dietary iron intake would be expected to be more susceptible to 2,3,7,8-TCDD toxicity than persons with a marginal iron intake. Similarly, females might be less vulnerable to 2,3,7,8-TCDD toxicity than males, since they usually store less iron in their bodies (33). This may explain the variation in toxicity levels among different species.

The most notable cases of human exposure to 2,3,7,8-TCDD are those associated with accidental releases in chemical factories or contact with contaminated materials/areas (33). The immediate symptoms of exposure to dioxins include a burning sensation in the eyes, nose, and throat, followed by headaches, dizziness, nausea, and vomiting. Several days later, severe itching, redness, and swelling of the face (more marked over the eyelids, nose, and lips) may develop just prior to the onset of chloracne. It has been shown that as little as 20 ug of 2,3,7,8-TCDD on the skin can lead to chloracne (33). Within the initial weeks after exposure, inflamed nodules as well as pustules appear on the face, forearms, shoulders, neck, and trunk, leading to comedones and cysts (33, 108, 130). Sometimes chloracne is preceded by erythematous and edematous skin lesions (108). After 1 or 2 months, acneform eruptions emerge, and the skin becomes hyperpigmented. At approximately the same time, muscles, primarily in the thighs and chest area, become aggravated upon exertion. Insomnia, extreme fatigue, nervousness, irritability, and the loss of libido also occur during this period (33, 108, 118, 130).

Other symptoms of exposure include arthralgias, porphyria cutanea tarda, hyperlipidemia, cutaneous hyperpigmentation, and hirsutism. Porphyria cutanea tarda, a skin condition that usually occurs as a photosensitive dermatosis, is caused by the development of vesiculobullous lesions over exposed areas (33). The dermatosis is precipitated by minor trauma, and may result in areas of healed bullae, crusts, scars, and milia (33). Blurred vision and the loss of taste and smell may also occur (33).

Patients with chloracne may exhibit increases in serum triglycerides and cholesterol. Lipids on the skin of such patients contain an increased proportion of cholesterol, squalene, and wax esters. Since the latter two substances are known to induce comedo formation in experimental animals, it is possible that the disturbance of lipid metabolism in cells of the sebaceous follicles is responsible for the dermal toxicity of 2,3,7,8-TCDD (116).

No fetal damage has been substantiated to indicate a mutagenic, teratogenic, or embryotoxic effect of 2,3,7,8-TCDD exposure in vivo (33, 132). However, chromosomal abnormalities have been reported in in vitro cytogenetic studies on human lymphocytes exposed to 2,4,5-T containing 0.09 ppm 2,3,7,8-TCDD (33). Chromatid breaks and deletions increased with increasing concentrations of 2,4,5-T. It was not possible to determine whether this was a toxic or genetic effect (33).

The clinical appearance of contaminated animals and the variety of lesions found during post-mortem examinations are identical for TCDFs, 2,3,7,8-TCDDs, and PCBs (107, 128). The toxicity of TCDF is approximately 10 percent that of 2,3,7,8-TCDD, and 1,000 times that of Aroclor 1242 (128). The correlation of metabolism with detoxification indicates that the greater toxicity of 2,3,7,8-TCDD versus TCDF could be the result of the relatively slower metabolism and excretion of 2,3,7,8-TCDD (120, 133). Studies indicate that TCDF metabolism is carried out through a detoxification mechanism; species that are able to metabolize and purge TCDF are more resistant to its acute toxicity. The multiple-organ and histologic damage may be a result of both the relative rate of formation and the further inactivation of the reactive metabolites in different organs (e.g., liver, kidneys, stomach, etc.).

Analyses of liver, adipose tissue, and blood samples of Yusho patients from both Japan and Taiwan indicated that the highest concentrations of PCDFs were found in the liver (Table 6-3). PCDF isomers present in the rice oil but not detected in the Yusho patients were 2,3,6,7-tetra-, 2,3,4,6,7-penta-, 1,2,6,7,8-penta-, 1,2,3, 4,8-penta-, and 1,2,3,4,6,7-hexa-CDFs (Figure 6-2). These PCDF isomers, which were metabolized and excreted fairly rapidly, have two vicinal unchlorinated C-atoms in at least one of the two aromatic rings in the PCDF nucleus (Figure 6-2) (79, 134). In contrast, PCDF isomers retained in the bodies, such as 2,3,6,8-tetra-, 2,3,7,8-tetra-, 1,2,4,7,8-penta-, 2,3,4,7,8-penta-, 1,2,3,7,8-penta-, 1,2,3,6,7,8-hexa-, and 1,2,3,4,7,8-hexa-CDFs, had lateral positions substituted with chlorine (Figure 6-2) (79, 134). The complete elimination of this compound from the organism is a function of the body's ability to metabolize it to polar metabolites, which can be conjugated and excreted.

Table 6-3

LEVELS OF PCDFs IN SAMPLES FROM YUSHO PATIENTS (79, 134)

	Liver		Adipose		Blood	
Isomer	Yusho, Japan	Yusho, Taiwan	Yusho, Japan	Yusho, Taiwan	Yusho, Japan	Yusho, Taiwan
2,3,6,8-Tetra-CDF	<0.005-0.7	ND*	<0.005-0.6	ND	ND	ND
2,3,7,8-Tetra-CDF	<0.005-0.3	0 060	<0.005-0.3	0.014	<0 003	<0 03
1,2,3,7,8-Penta-CDF	ND	0.194	ND	0.044	ND	0 02-0 03
1,2,4,7,8-Penta-CDF	<0.005-7.1	0.042	<0.005-0.8	0.014	ND	0 04-0 06
2,3,4,7,8-Penta-CDF	<0.005-6.9	0.091	<0.005-5.7	0.068	0 003	0 08-0 120
1,2,3,4,7,8-Hexa-CDF	<0.005-2.6	0.193	<0.005-0.5	0.088	<0.006	0.06-0 150

* ng/g.

** ND = Not detected.

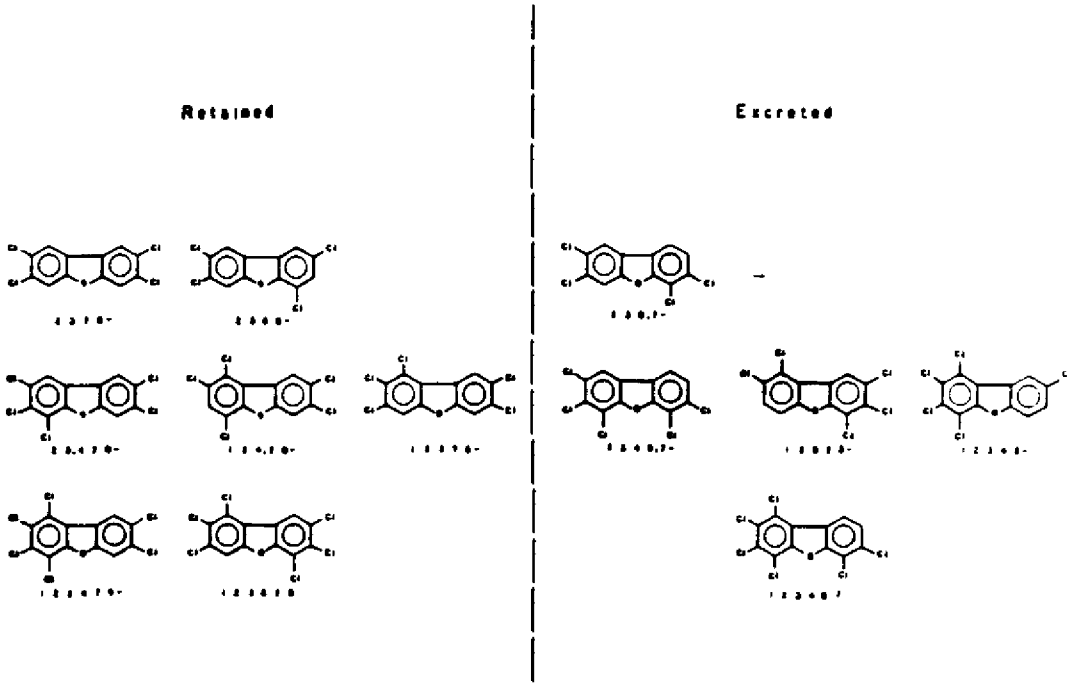

Figure 6-2. PCDF Isomers Retained and Excreted in Yusho Patients

Section 7

CHEMICAL AND BIOASSAY ANALYSES

CHEMICAL ANALYSES

The toxicities of some PCDD and PCDF isomers, which have been demonstrated by tests (Section 6) and, to some extent, by accidental exposures of humans to these compounds (Section 2), have prompted extensive efforts to quantitatively measure these chemicals in various chemical, biological, and environmental media (36, 136). These toxicity considerations have further dictated the requirement to analyze for these chemicals at concentrations ranging as low as parts-per-trillion (10^{-12} g or picograms of analyte per gram of sample). Development of an entirely new realm of analytical chemical capability has been required to achieve reliable quantitative determinations at the part-per-trillion level; this has only recently been successfully realized. Even when the sample medium contains higher concentrations of PCDDs/PCDFs, analysis for these compounds can be a formidable task because of the large numbers and quantities of other chlorinated hydrocarbons which are frequently present in many samples of interest. These other hydrocarbons can complicate or even preclude accurate determination of the PCDDs/PCDFs if appropriate analytical methodology is not utilized.

In the present survey, the current status of analytical procedures for the determination of PCDDs/PCDFs is reviewed. A summary of the instrumental methods which have been used for the detection and/or quantification of PCDDs/PCDFs is given in Table 7-1. While the focus of this survey is primarily aimed at techniques applicable to PCB-containing fluids used in electrical devices, it is desirable to discuss the evolution of such analytical methods in a somewhat historical sense, particularly since most of the reported analytical procedures have been designed for other types of sample media.

SCOPE OF THE ANALYTICAL PROBLEM

As discussed earlier, there are 75 PCDD isomers and 135 PCDF isomers. The magnitude of the analytical task associated with the determination of PCDDs/PCDFs in complex samples thus becomes apparent, especially when one recognizes the difficulty of separating and identifying a large number of isomers within a given chlorinated

Table 7-1

INSTRUMENTAL METHODS APPLIED TO DETECT PCDDs/PCDFs IN SAMPLE EXTRACTS

Method	Application	Reference
Screening/Qualitative Analyses		
UV Spectroscopy	TCDD	137
Thin-Layer Chromatography	Fats and oils	138
Electron-Capture Gas Chromatography (EC/GC)	PCDDs in herbicide formulations, chlorophenols, TCDDs	139, 140, 141, 142
Probe-Mass Spectrometry (MS)	Analysis of milk, fish, biological tissue	143
Thermally Modulated Electron Capture Gas Chromatography (TMECD)	Biological tissues, fat, TCDD, naphthalene, chlorine, benzopyrene, anthralates, pesticides, TCDD and PAH standards	144
Highly Specific/Quantitative Analysis		
Packed-Column GC-Low Resolution MS (Selected Ion Monitoring)	Chlorinated phenols - TCDD	145
	Pentachlorophenol - TCDD	146
	Pentachlorophenol - TCDD	147
	Herbicide Orange formulations - TCDD	148
	Surface wipes and metal surfaces - TCDD	149
Packed-Column GC-High Resolution MS (Selected Ion Monitoring)	Beef adipose and liver - TCDD, HxCDD, HpCDD, OCDD	150
	Chemical wastes, aqueous effluents, soils - TCDD	151
	Biological tissue, plant materials - TCDD	152
	Beef adipose - TCDD	136
Capillary-Column GC-High Resolution MS (Selected Ion Monitoring)	Human milk, beef liver, fish, water, sediment - TCDD	153
	Particulates from combustion sources - TCDD isomers	154
Capillary-Column GC-Low Resolution MS (Selected Ion Monitoring)	Separation of TCDD, PCDD, HxCDD isomers, HpCDD, OcCDD, PCDF, PCDDs in fly ash	155, 156, 136, 157, 10
	PCDFs in oils, PCBs, fly ash and pyrolysis products, human tissue	53, 156, 150
	PCDDs and PCDFs in incinerator effluents	136
	PCDDs and PCDFs in human tissue	79
Capillary-Column GC	Combustion effluents - PCDDs	160
	Human samples - PCDDs, PCDFs	79, 135
Liquid Chromatography Packed Column GC-Low Resolution MS (Selected Ion Monitoring)	TCDD, HxCDD, HpCDD, and OCDD isomers in particulates, isomer separation and identification	136, 161
GC-Negative Chemical Ionization MS	Animal tissue and wood - PCDDs	162
	Human blood and tissue, fly ash - PCDDs	79, 163, 135
GC-Negative Ion MS	PCDFs in fish and sediments	163, 164
GC-Atmospheric Pressure Ionization MS	Identification of TCDD isomers in standard mixture, fish	142

class, all of which are quite similar structurally, and thus similar in chemical and physical properties. This task is further complicated by the fact that many of the isomers of the PCDDs/PCDFs have not been prepared in pure form, and thus are not available for use in developing and calibrating analytical methods.

Several synthetic routes for preparing PCDDs (137, 165, 166, 167, 168, 169, 170) and PCDFs (45, 48, 68, 167, 171, 172, 173) have been reported, and various PCDD and PCDF isomers have been produced by these reactions. The entire sets of the isomers of tetra-, hexa-, and hepta-CDDs, as well as octa-CDD, have been synthesized (161, 169, 170), as have 2 di-, 4 tri-, and 14 penta-CDDs (34, 174, C. Rappe, Personal Communication). All 38 tetra-CDF isomers (175) and some of the penta-CDF isomers (174) have been synthesized.

The PCDD and PCDF isomers prepared by the various investigators cited have been characterized using several methods, including gas and liquid chrometography, mass spectrometry, proton- and ^{13}C-NMR, and photolytic dechlorination coupled with pattern recognition techniques (136). However, few, if any (except 2,3,7,8-TCDD), have been structurally confirmed by single-crystal X-ray diffraction, which is the most definitive method. This is due in part to the fact that only quite small quantities of the synthesized isomers have been available; in addition, single-crystal X-ray diffraction requires considerable time and expense.

The PCDD and PCDF isomers prepared by these investigators have not been widely available to laboratories seeking these compounds for use as analytical standards. However, a limited number of PCDD and PCDF isomers, including several ^{13}C- and ^{37}Cl-labelled compounds, have been prepared in sufficient quantities for sale to numerous investigators by a commercial synthetic laboratory (KOR Isotopes, Cambridge, Massachusetts). PCDD standards are available only as mixtures; PCDF standards have been synthesized and isolated as individual isomers. Even in those laboratories which have synthesized or acquired PCDD and PCDF standards, there are no reported instances in which the quantities of these compounds have been sufficient to thoroughly evaluate and calibrate the complex analytical procedures for entire sets of isomers of several chlorinated PCDD and PCDF classes.

In spite of these limitations, there has been widespread interest in the determination of specific PCDD and PCDF isomers. This interest stems largely from the fact that the toxicity of these compounds is markedly dependent on both the number and position of the chlorine substituents.

The toxicology of PCDDs and PCDFs is discussed more extensively in Section 6. It is clear from that discussion that there is a need to perform isomer-specific analyses of PCDDs and PCDFs in many types of sample media, and that simply determining these compounds by chlorinated class does not generally provide sufficient information to adequately evaluate hazards. However, the extent to which such analyses can currently be accomplished is still rather limited for the reasons discussed above.

Many of the early analytical studies focused on the determination of 2,3,7,8-TCDD in various biological and environmental samples, because of the extreme toxicity of 2,3,7,8-TCDD, which has been associated primarily with 2,4,5-trichlorophenol and certain herbicide derivatives (2,4,5-T) of this chlorophenol (all widely used at one time). In addition, this PCDD isomer was one of the earliest available in sufficient quantity to use as an analytical standard. Much of the currently extant analytical methodology for PCDDs and PCDFs thus stems from that developed originally to detect 2,3,7,8-TCDD. Some of this methodology was gradually adapted, modified, and expanded to accommodate analyses of other PCDDs and PCDFs, although entirely new methods have also been developed. Among the many types of samples which have been analyzed for PCDDs and/or PCDFs are the following (36, 136):

- Animal tissues - adipose, muscle, liver, blood, whole animal (bovine, fish, deer, elk, turtle, rat, rabbit, birds).
- Human tissues - blood, adipose, liver, kidney.
- Human and animal milk.
- Plant material.
- Soils and sediments.
- Water - rivers, streams and lakes, wells, leachates.
- Chemical formulations and wastes (2,4-D; 2,4,5-T, PCBs, chlorophenols, diphenylether herbicides, and other herbicides and industrial chemicals).
- Combustion products - fly ash, bottom ash, soot, condensate particulates and other stack effluents, water and other process fluids from combustion of municipal waste, coal, wood, chemical wastes, PCBs, PCB-treated materials and wastes.
- Industrial hygiene samples - wipes, air samples (solid sorbents).
- Hazardous waste site samples - complex chemical mixtures, landfill sludges, leachates.

It should be mentioned that the level of sophistication applied in these analyses, and the objectives thereof varied widely. In some cases, the objectives have been primarily to screen samples (qualitatively or semiquantitatively) to identify chlorinated classes of PCDDs and/or PCDFs. In other instances, the objective of the analysis has been to accurately quantify specific PCDD and/or PCDF isomers in such samples. Both types of analyses can be useful, depending on the purposes for which the analytical results are to be used. The most specific of these methods employs mass spectrometry (MS). Prerequisites for best analyses include representative sampling, good storage, efficient extraction, and sample purification (containment) followed by good separation, ultrasensitive detection and quantification, and most desirable confirmation (113).

CRITERIA FOR SELECTING ANALYTICAL PROCEDURES

There are several criteria which may be used to assess the efficacy of specific analytical procedures for the determination of PCDDs/PCDFs, depending upon the objectives of the analysis. These criteria are discussed below.

A standard practice applied by most competent laboratories which perform analyses for PCDDs/PCDFs involves the addition of isotopically labelled PCDD and/or PCDF isomers to the sample in known quantity prior to analysis, and determination of these labelled compounds along with the native or unlabelled PCDDs or PCDFs. The percent recovery of the isotopically labelled standard provides a measure of the efficacy of the analytical procedures. Typically, ^{37}Cl-labelled or ^{13}C-labelled PCDDs or PCDFs have been used for this purpose. However, since only a few such standards have been prepared and are generally available (certain labelled isomers of the tetra-, hepta-, and octa-CDDs, and labelled 2,3,7,8-TCDF are commercially available), the extent to which these procedures can be utilized is limited. The addition of a labelled PCDD/PCDF to a sample is also useful in that the added compound can be utilized as a true internal standard for purposes of quantitating the native PCDDs/PCDFs in the sample. Such an approach presumes that losses of the added internal standard incurred in the course of processing the sample are proportionately the same as the losses of the native PCDDs/PCDFs in the sample. Since the internal standard is added in an accurately known quantity, however, by measuring the native compound relative to the labelled internal standard, it is possible to reliably quantitate the native compound in the sample.

Since PCDDs/PCDFs have been detected at concentrations spanning a relatively wide range in various sample matrices (typically parts-per-trillion to parts-per-thousand), it is desirable to utilize analytical methodology which can detect (and if desired, quantitatively measure) PCDDs/PCDFs over this entire range.

If large numbers of samples are to be analyzed, as is frequently the case in environmental assessments, the analytical methodology should be amenable to efficient and reliable processing of such large numbers of samples in a reasonable time period. The complexity of reliable analytical procedures for determining PCDDs/PCDFs is such that even the best methods currently available are quite labor- and resource-intensive. These analyses are therefore quite expensive, and single analysis often requires 1 to 2 days, although economies can obviously be realized by processing samples in batches.

To the extent possible, acceptable analytical procedures for determining PCDDs/PCDFs should be applicable to the analysis of a wide variety of different sample matrices. As shown in Tables 7-1 and 7-2, the procedures for separation and instrumental detection which have been developed for PCDD/PCDF analyses are rather similar for many types of samples.

SAMPLING METHODS

The procedures utilized in obtaining a sample to be analyzed for PCDDs/PCDFs can markedly affect the results obtained. A detailed discussion of sampling methods is beyond the scope of this review, but it should be noted that this aspect of PCDD/PCDF analysis is perhaps one of the most neglected areas. In general, the dictates of good analytical practice should be applied in sampling: homogeneity of the sample matrix; collection of a truly representative sample; statistical considerations with respect to replicate analyses; and the resultant effects on precision and accuracy. In cases where samples are acquired by simple grab sampling, such considerations can usually be applied. In other instances (e.g., collection of flue gas samples from incineration sources or surface wipe tests), the sampling procedures are far less standardized, and their effectiveness and limitations have not been adequately evaluated.

Sampling methodology is discussed in a recent Canadian Government report on analytical methodology applicable to PCDDs (185). A discussion of some of the sampling

procedures applicable for collection of flue gas samples has been presented by Tiernan, et al. (160), and a detailed discussion of environmental sampling requirements and guidelines has been prepared by the American Chemical Society (186).

Table 7-2

LIST OF EXTRACTION AND CLEANUP PROCEDURES FREQUENTLY USED FOR PCDD/PCDF ANALYSES

Originator	Procedure/Reference	Application
U.S. Environmental Protection Agency	Acid extraction (153)	Fish; adipose tissue; milk, water; soil; sediments
	Neutral extraction (153)	Fish
U.S. Food and Drug Administration	(176)	Chemical formulations, fish
U.S. Fish and Wildlife Service	(177)	Fish
Dow Chemical Company	Acid extraction (178, 179)	Fish; milk
	Neutral extraction (180)	Fly ash; dust, urban particulates; municipal sludge, soil
Wright State University-Brenm Laboratory	(180, 181)	Soil, chemical waste; aqueous effluents
	(160, 182)	Particulates and other samples from flue gas stack
Umea University, Sweden	(10)	PCBs
	(3, 183, 184)	Fly ash, soot, wipe tests
	(79, 135)	Blood

SAMPLE EXTRACTION, PURIFICATION, AND SEPARATION

Analytical methods which have been developed for determination of PCDDs/PCDFs in various sample matrices generally entail a sequence of three operations:

- Extraction of PCDDs/PCDFs from the sample matrix, which in some cases involves digestion or destruction of the matrix.

- Preliminary separation of PCDDs/PCDFs from major matrix constituents and other major chlorinated residues and chemical contaminants.
- Quantification and confirmation of PCDDs/PCDFs in the cleaned-up sample extract.

Procedures to accomplish these operations have been developed and applied in several different laboratories. These procedures are similar in many respects, although they differ in various details, such as the choice of liquid chromatography columns, and the digestion or extraction reagents. A representative listing of several methods that are used by major laboratories currently performing PCDD and/or PCDF analysis is presented in Appendix D. Other reported variations of such methods are discussed and evaluated in a recent Canadian report (185).

All of the methods listed in Appendix D entail initial addition of internal standards, followed by destruction of the sample matrix (where practical) or extraction with an organic solvent, partitioning of the PCDDs/PCDFs into an organic phase and polar constituents into an aqueous phase and discarding of the latter, and liquid chromatographic cleanup (and in some cases HPLC cleanup) of the extracts. The cleanup steps are intended to remove matrix components, such as lipids, as well as most of the chlorinated hydrocarbon residues in the sample (e.g., PCBs, DDE, other common chlorocarbon residues, and fused-ring aromatic compounds), which may interfere with subsequent detection and quantitation of PCDDs/PCDFs. Albro and Parker have recently discussed the types of chlorinated hydrocarbons which may constitute interferences, and fractionation procedures aimed at elimination of these (74). Some of the more frequently used extraction and cleanup procedures are listed in Table 7-2, followed by a discussion of the efficacy of extraction and cleanup methodology.

EFFICACY OF EXTRACTION AND CLEANUP METHODOLOGY FOR PCDDs/PCDFs

The efficacy of extraction and cleanup methodology for determining PCDDs/PCDFs is generally gauged by the extent of recovery of the initially added labelled internal standards. This has been applied less frequently for PCDF determinations, because only one labelled PCDF has been available ($^{37}Cl_4$-2,3,7,8-TCDF). Collaborative testing of some of these methods has been accomplished through EPA's Dioxin Implementation Plan (180). Few of the results of these studies have yet been published. The FDA also conducted a limited assessment of several of these methods for fish analyses (187). Most of the methods cited seem to be relatively effective in so far as they have been tested, when accomplished by skilled and experienced analysts.

One type of sample which has received particular attention in terms of evaluating the efficiencies of extracting PCDDs/PCDFs using various procedures is particulates (such as fly ash). Lustenhouwer, Olie, and Hutzinger (60) have reported the results of extracting aliquots of the same fly ash sample using six different extraction methods, which have been used by various investigators who have reported data on determinations of PCDDs/PCDFs in the literature. According to these results (Table 7-3), Soxhlet extraction with toluene (following acid treatment) was the most effective of the several procedures evaluated.

More detailed subsequent studies from the same laboratory in which still other procedures were evaluated led to the conclusion that Soxhlet extraction with benzene is approximately equally effective, and far superior to simpler extraction methods (378). In the latter study, the effect of the period of extraction, ranging from 24 to 72 hours, was also evaluated. The results of these tests, shown in Table 7-4, indicate that a 24-hour extraction period was sufficient to remove the bulk of the PCDD/PCDF constituents. Cutie (153) has also reported results of similar studies on the extraction of 2,3,7,8-TCDD from fly ash, soil, and active carbon.

There have been comparatively few measurements of PCDDs/PCDFs in electrical equipment PCB fluids (see Section 3). As such, methodology for this purpose has not been extensively reported.

The extraction and cleanup procedures applied in several of these studies were generally similar to those described earlier in this section for determinations of PCDDs/PCDFs in other samples. The most detailed accounts of such procedures applicable to PCB electrical fluids are those given by Buser (156), Rappe, et al (87), and Albro and Parker (74). Polychlorinated diphenylethers, which are also present in some fluids of this type, can give rise to mass spectral fragment ions, which coincide with those monitored as indicators of the CDFs (74). It is thus important that the chlorinated diphenylethers be effectively removed from the extracts by separation techniques prior to GC/MS analysis. Adequate separations can be achieved by careful attention to the elution procedures used in the liquid chromatographic fractionation sequence (74).

INSTRUMENTAL METHODS

Table 7-1 lists the instrumental methods which have been applied for detection and/or quantification of PCDDs/PCDFs in sample extracts prepared by the procedures given in Table 7-2 and Appendix D. Table 7-1 shows the types of analytical applications

Table 7-3

EXTRACTION EFFICIENCIES OF POLYCHLORINATED COMPOUNDS FROM FLY ASH (60)

Method	Analytical Results (Total, ppb) Chlorophenol		PCDD		PCDF	
1. Toluene, acid treated, Soxhlet extraction	1364	100%	1309	100%	1407	100%
2. CH_2Cl_2, shaking treatment	240	18	124	1	23.5	2
3. Acetone/hexane, ultrasonic treatment	92	7	No	1	No	1
4. CH_2Cl_2, Soxhlet extraction	434	32	81.8	6	155	11
5. $CHCl_3$, Soxhlet extraction	592	44	168	13	260	20
6. Toluene extraction of solution of fly ash in 48% HF	446	33	113	9	230	16

Description of methods:

1. Fly ash (25 g) is stirred in 1N hydrochloric acid (200 ml). After centrifugation, the residual fly ash is washed with deionized water over a Buchner funnel and dried at room temperature. The fly ash is extracted for 35 hours with toluene using a Soxhlet apparatus.

2,3. Fly ash (10 g) is shaken manually (Method 2) or treated in an ultrasonic bath for 5 minutes (Method 3) in the solvent indicated. For both methods, this is repeated four times. The extracts are combined for cleanup and analysis.

4,5. Fly ash (25 g) is extracted for 35 hours, using a Soxhlet apparatus.

6. Fly ash (5 g) is dissolved in a 48% HF solution. The solution is then extracted with toluene. Some particulate matter remained.

(e.g., screening or qualitative detection, quantitative analysis, isomer determinations) for which these various methods have been applied, along with examples of reports of such applications. As indicated in Table 7-1, several methods have been developed for rapid screening of samples for the presence of PCDDs/PCDFs, such as UV-spectroscopy, thin-layer chromatography. For example, gas chromatography using an electron capture detector (GC/ECD) offers the sensitivity needed for such a screening procedure, but does not have the specificity that is needed to eliminate false positives. The ECD responds to any molecule that has an affinity for capturing electrons in an ionized field. This includes molecules containing halogens (e.g., pesticides), oxygen (e.g., phthalate esters, ethers, ketones, etc.), and other hetero atoms, such as sulfur and nitrogen (144).

Table 7-4

INFLUENCE OF TIME ON EXTRACTION EFFICIENCY
FOR PCDDs AND PCDFs* (188)

	PCDD (%)					
	TCDD	P_5	HCDD	H_7CDD	OCDD	Average
1st 24 hours	97.0	97.4	98.4	99.3	99.5	98.0
2nd 24 hours	2.3	2.0	1.3	0.7	0.5	1.6
3rd 24 hours	0.7	0.6	0.3	0.1	0.0	0.3

	PCDF (%)					
	TCDF	P_5	HCDF	H_7CDF	OCDF	Average
1st 24 hours	97.7	98.4	98.6	99.4	99.5	98.5
2nd 24 hours	1.7	1.3	1.1	0.6	0.5	1.2
3rd 24 hours	0.8	0.4	0.3	0.1	0.0	0.3

* Sample pretreatment with 1N HCl; Soxhlet extraction with toluene.

Radian Corporation has developed a prototype detector system for gas chromatography, called a thermally modulated electron capture detector (TMECD) system. This system consists of two electron capture detectors in series with a temperature-controlled

catalytic pyrolysis unit between them. Different classes of organic compounds react to the pyrolysis conditions in different ways, resulting either in an enhanced signal, a diminished signal, or no change in the magnitude of the signal. The ratio of the peak area measured for the detector after the reactor to that for the detector prior to the reactor is a property characteristic of each compound. This peak area ratio is used in conjunction with its retention time to increase the confidence level of the identity of a given compound.

In addition to this method (which is still under development and needs validation), several techniques have been developed which are dependent upon biological responses to the toxic PCDDs/ PCDFs. These bioassay techniques are discussed separately in this section.

It is generally accepted that the only technique which has both the required sensitivity and specificity for reliable detection and quantitation of PCDDs/PCDFs is coupled gas chromatography-mass spectrometry (GC/MS). Mass spectrometric analysis for PCDDs/PCDFs is based on the presumption that these compounds exhibit specific mass spectral fragmentation patterns. The electron impact mass spectra of PCDDs and PCDFs exhibit intense molecular ions (M^+) with the appropriate ion clustering, which is due to the two chlorine isotopes. PCDDs mainly fragment to M^+-COCl and M^+-2COCl ions. Smaller intensities of various other ions appear in the spectra of both types of standards, thus enabling reliable analysis of PCDDs/PCDFs (189).

In most instances, monitoring the molecular ions of the PCDDs and PCDFs will easily distinguish these compounds from other chlorinated aromatic hydrocarbons, which may be present in the sample extracts. However, some PCBs, as well as DDE, can fragment to yield ion masses which may interfere with the detection of certain PCDDs, unless rather high mass spectral resolution is employed. Also, the identification of PCDFs may be complicated by the presence of chlorinated diphenylethers, since the latter compounds yield intense M^+-Cl_2 ions, which have the same exact mass and number of chlorine atoms as the corresponding PCDF (156). In such cases, successful analysis is dependent upon the ability of the preseparation procedures and/or the gas chromatographic fractionation to separate such interfering compounds from the PCDDs/PCDFs before the latter enters the mass spectrometer. While it is desirable to obtain complete mass spectral scans in GC/MS analysis in order to identify the analytes of interest, this is not possible if very low detection limits (e.g., part-per-trillion) are needed. In the latter case, which is the more common requirement in analyzing environmental samples, only a few ions in the mass spectra of the PCDDs/PCDFs

of interest are typically monitored, utilizing a technique termed Selected-Ion Monitoring. This is discussed further below.

Earlier analyses of PCDDs were aimed primarily at determining the low levels of TCDD, and utilized less sophisticated gas chromatographic techniques than those presently available. The first attempt to apply mass spectrometry for quantitative determination of TCDD was the high-resolution mass spectrometric technique reported by Baughman and Meselson (143). This technique did not utilize prior gas chromatographic separation at all, the sample extract was introduced via a direct inlet probe. These investigators modified a high-resolution mass spectrometer to permit repetitive high-resolution scanning over a narrow mass range (typically 0.3 AMU) at a rapid rate (4 scans/second). Mass spectrometric signals were acquired using a signal-averaging computer, which resulted in sufficient sensitivity (approximately 1 picogram of injected TCDD).

Due to the relatively high mass spectral resolution achieved, this technique was adequate for the purposes of distinguishing the molecular ion of TCDD from ions arising from the sample matrix (mostly biological samples). However, it was soon realized that the TCDD ions could not be distinguished from those originating from certain PCBs, if the latter were present in the sample extract in large concentrations relative to the TCDD (part-per-million versus part-per-trillion). Another limitation of the latter method was its inability to separate TCDD isomers; the mass spectrometric response observed with this method was obviously attributable to any TCDD isomers in the sample, or any compounds given a TCDD fragment, not just the 2,3,7,8-TCDD which was of major interest.

During the same period that the Baughman-Meselson method was being applied, several investigators reported the use of coupled gas chromatography-low resolution mass spectrometry for determinations of TCDDs in chlorinated phenols and 2,4,5-T herbicide formulations (145-149). These studies utilized open-tubular packed gas chromatographic columns, which were capable of separating TCDD from other PCDDs of different chlorine content, but had little capacity for separating discrete TCDD isomers. The detection limits achieved in these determinations were in the range of 0.001 to 0.1 ppm. These methods derived their specificity from the cleanup methods used. Furthermore, these methods required that the analyte exhibit the appropriate GC retention time and a mass spectral response at the appropriate ion masses corresponding to TCDD.

Due to concerns over the possible accumulation of 2,3,7,8-TCDD in the food chain (from the use of 2,4,5-T on cattle-grazing land), the U.S. EPA initiated the Dioxin Implementation Plan (DIP) in 1975. This program was intended to quantitatively assess the levels of 2,3,7,8-TCDD in beef tissues and related environmental samples. The program prompted the development of combined GC (packed-column)-high resolution mass spectrometric techniques for determining TCDD at four different laboratories i.e., Wright State University, University of Nebraska, EPA/RTP, and Dow Chemical Company (136, 150, 152, 181). The mass spectrometric methods were similar to methods applied by Baughman and Meselson (143), but were superior in that high-resolution scanning was generally accomplished over a narrower mass range and at a more rapid scan rate. This improved both the resolution capability and the sensitivity of the technique. Wright State's adaptation of this procedure permitted as many as four ions to be monitored.

A further enhancement of the capabilities of the GC-high resolution mass spectrometric technique for analysis of PCDDs/PCDFs entailed the incorporation of capillary GC columns (wall-coated open-tubular columns). This has been accomplished at both the EPA/RTP (153) and Wright State laboratories (154). The use of capillary GC columns provided the capability to separate discrete isomers of the PCDDs/PCDFs; until recently, few of these have been available in pure form. The use of capillary column GC-high resolution MS also further improved the ability to obtain accurate quantitative data on the low concentrations of PCDDs/PCDFs in environmental samples. Typical results of this highly specific technique are shown in Table 7-5.

Due to patent problems in the United States, the technique of capillary columns was developed and extensively used in Europe. Investigations of the use of capillary GC columns for the separation of PCDD isomers were undertaken as early as 1975 by Buser (155), who prepared eight HxCDD isomers and demonstrated that these could be separated on a column having about 35,000 theoretical plates, using a temperature program which increased the column temperature by 2°C/minute. Subsequently, Buser (155) demonstrated separations of nine TCDD isomers available at that time. More recently, Buser (155) and Buser and Rappe (157) have reported the results of separating all 22 TCDD isomers on three different capillary columns.

Other capillary columns have also been tested for separating the 22 TCDD isomers, such as new hybrid columns coated with stationary phases OV-17 and/or Silar-10C (Figures C-1 and C-2, Appendix C). It should be noted that these techniques provided for the separation of the highly toxic 2,3,7,8-tetra-CDD isomer from the other

Table 7-5

TYPICAL ANALYTICAL RESULTS FOR QUALITY ASSURANCE SAMPLES. ANALYSIS OF FISH FOR 2,3,7,8-TCDD (153)

Sample* (g)	^{37}Cl-TCDD Recovery** (%)	TCDD Detection Limit (ppt)#	TCDD Detected (ppt)#	TCDD Fortification Level	
				pg	ppt
5(1)#	62	2	20	110	22
5(1)	52	4	34	185	35
5(1)	82	3	ND	0	0
5(4)	100	3	ND	0	0
5(3)	54	1	19	70	14
5(3)	100	2	ND	0	0
5(3)	78	2	ND	0	0
5(1)	92	2	19	55	11
5(1)	97	5	45	240	48
10(1)	100+	1	8	130	13
10(1)	100+	4	43	600	60
10(2)	100+	7	ND	0	0
10(2)	100+	3	ND	0	0
10(1)	100+	4	76	1250	125
10(4)	67	1	ND	0	0
10(1)	93	3	56	650	65
10(1)	84	4	73	620	62

* (1) Ocean perch, (2) lake trout, (3) beef liver, and (4) method blank.

** Each sample has been fortified with 5 or 10 ng ^{37}Cl-TCDD.

Corrected for % recovery losses; ND = not detected; ^{37}Cl-TCDD mean % recovery, 86%; TCDD mean % accuracy, ±15%.

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21 isomers. Such capillary columns also provided the means for separating penta- and hepta-CDD isomers (Figure C-3), as well as tri-, tetra-, penta-, and hexa-CDFs (Figures C-4, C-5, and C-6).

Capillary column GC-low resolution mass spectrometry is being increasingly utilized at several laboratories to provide data on a wide range of PCDD/PCDF isomers in various types of samples (10, 53, 136, 155, 157, 158, 159) including PCB electrical fluids. A summary of the quantification of separate PCDD and PCDF isomers in samples from PCB fires and incinerator fly ash is given in Table 7-6.

Dow Chemical scientists have recently achieved considerable success in the separation of tetra- and hexa-CDD isomers by HPLC fractionation of discrete isomeric compounds (136, 161). This technique appears to be particularly powerful when used in conjunction with GC/MS techniques, and does not necessitate the use of capillary GC columns. No data on separation of PCDF isomers by this method has been reported.

Several newer mass spectrometric techniques have also been recently investigated for the analysis of PCDDs/PCDFs. Among these are negative-ion mass spectrometry (163), negative-ion chemical ionization mass spectrometry (162), and atmospheric pressure-ionization mass spectrometry (142). Results obtained thus far show that all of these offer enhanced sensitivity, selectivity, and specificity. For furan isomers in particular, there is a dramatic increase in sensitivity using negative-ion chemical ionization MS as compared to normal electron impact MS.

QUANTIFICATION

To date, quantification has been based on peak area measurements by comparison with either internal standards or a calibration curve of known quantities of authentic standards (external standards). It has generally been assumed that all isomers of a particular PCDD or PCDF have the same response using the MS quantification. Recently, Gara, et al. (189), and Rappe, et al. (113), have studied responses of 28 and 20 different PCDF isomers, respectively. Findings of both studies showed a 3-fold variation in the response, using the electron impact (EI) mode, and up to a 20-fold and 25-fold variation, respectively, using the negative chemical ionization (NCI) mode. For the higher chlorinated homologues, the differences appeared to be much smaller.

Table 7-6

QUANTITATIVE ANALYSES OF PCDF AND PCDD ISOMERS IN SAMPLES FROM PCB INCIDENTS AND INCINERATOR FLY ASH

Isomer	Binghamton* (ppm)	Norrtalje** (ug/g)	Skovdal (ug/m²)	Surahammar# (ug/m²)	Municipal Waste (ppb)† Fly Ash	Municipal Waste (ppb)† Particulate	Industrial Fly Ash** (ppb)	Municipal Fly Ash (ppb)
Mono-CDF	-	20-43	-	-	-	-	-	-
Di-CDF	-	15-43	-	-	-	-	-	-
Tri-CDF	-	5-14	-	-	-	-	-	-
Tetra-CDF	28	3-4	0.01-0.6	<0.02-0.0	111	460	180	9
Penta-CDF	670	-	0.1	<0.01-3.3	196	960	530	23
Hexa-CDF	965	-	0.06	<0.01-1.8	361	1600	350	50
Hepta-CDF	460	-	0.008	<0.015-1.5	177	1130	300	250
Octa-CDF	40	-	0.005	0.002-0.3	18	140	20	135
Tetra-CDD	1.2	-	-	-	154	100	400	3
Penta-CDD	5.0	-	-	-	182	800	1400	20
Hexa-CDD	4.7	-	-	-	326	1370	1000	50
Hepta-CDD	7.0	-	-	-	288	1370	300	180
Octa-CDD	2.0	-	-	-	106	310	150	210

* Reference 29.
** Reference 2.
# Reference 190.
† Reference 60.

CONFIRMATION

Confirmation of PCDDs/PCDFs can be based on the proper molecular ions and fragments, as well as the intensities of the ion clusters due to the Cl isotopes. Moreover, if a sample contains a multitude of PCDD/PCDF isomers, the positive identification of PCDDs/PCDFs could be confirmed by cluster analyses (113).

For adequate confirmation of PCDDs/PCDFs, there are several parameters available Compared to an authentic standard, the PCDDs/PCDFs in the sample should have the same following parameters: retention volume in the column chromatography in the cleanup steps, retention time in the gas chromatographic separation, molecular weight, fragmentation and chlorine cluster in the MS detection, and difference in response factors comparing the EI and the NCI mode. These parameters, in addition to access to well defined weighted standards, give the possibility of reliable analysis of PCDDs/PCDFs.

BIOASSAYS

Numerous clinical biomedical parameters have been shown to be altered in animals and humans receiving different doses of PCDDs, PCDFs, and PCBPs (Section 6). In a number of investigations, 2,3,7,8-TCDD served as a prototype of individual PCDDs and PCDF congeners and their mixtures, all of which produce a similar and characteristic pattern of toxic lesions, including hyperkeratosis and squamous metaplasia in the skin, and induction of a number of coordinately expressed enzymes through stereospecific, reversible binding to a cytosolic receptor protein (which has been identified in several mammalian species and in cell cultures).

As described in Section 6, the potencies of the biologic and toxic responses for halogenated aryl hydrocarbons are also dependent on structure. Maximal activity was observed for the 2,3,7,8-TCDD congener, which contains a chlorine substitution pattern at the four lateral positions of the dibenzo-p-dioxin molecule (191). Further Cl substituted at the 1, 4, 6, and 9 positions tends to diminish the activity, whereas the removal of one or more of the laterally substituted Cl atoms leads to a marked loss (or complete loss) of activity. The results of recent studies suggest that the initial event in the mechanism of action of 2,3,7,8-TCDD and related compounds is the reversible non-covalent binding to a receptor protein. There appears to be an excellent correlation between the relative toxicities of halogenated aryl hydrocarbons and their potencies as AHH inducers and their receptor protein binding avidities (191). This suggests that both the receptor binding and AHH induction

assays may be useful short-term in vitro bioassays for total PCDD/PCDF biological activity, which would in turn reflect potential toxic potency.

The significance of the bioassays, in addition to the assessment of the toxicological effects, is their usefulness as a rapid and reliable screen for detecting biologically active substances prior to chemical identification and quantification. The following discussion will focus on some of these bioassay techniques.

Aryl Hydrocarbon Hydroxylase (AHH) Induction

2,3,7,8-TCDD has been known to alter the activity of a number of enzyme systems, the most extensively studied being the hepatic microsomal monooxygenases. The monooxygenase enzyme system, consisting of NADPH-cytochrome P-450 reductase and cytochrome P-450, is an integral part of the smooth endoplasmic reticulum, and catalyzes the catabolism of lipophilic compounds to more polar metabolites.

The induction of the hepatic monooxygenases by 2,3,7,8-TCDD occurs in most tissues of most species. It should be noted that these bioassays are extremely sensitive to a variety of chlorinated aromatic hydrocarbons. A large number of specific enzymes have been assayed; the most extensively studied of them, AHH, is discussed below.

AHH activity is a measure of one cytochrome, cytochrome P-448. The induction of AHH is mediated by the reversible stereospecific binding of halogenated aromatic hydrocarbons to a cytosol protein, the cytosol induction receptor (192), which mediates the nuclear uptake of the toxicant (193). The receptor is determined by the Ah locus in mice (192). The Ah locus, and hence the cytosol receptor, controls not only the induction of AHH activity but also a battery of coordinately expressed enzymes (192).

Two lines of evidence suggest that toxic lesions produced by TCDD, as well as biochemical responses, are mediated by the cytosolic receptor. First, the binding affinities of a large number of halogenated aromatic hydrocarbons for the receptor correspond very closely with the rank order of their toxic potencies (193). Second, two toxic responses produced by TCDD in mice, thymic involution and cleft palate formation in fetuses, have been shown to segregate with the Ah locus (192).

Compared with classical polycyclic aromatic hydrocarbon inducers of AHH, such as 3-methylcholanthrene, 2,3,7,8-TCDD produces a parallel dose-response curve; both

compounds produce the same maximum response in AHH activity. However, 2,3,7,8-TCDD is 3 x 10^4 times more potent that 3-methylcholanthrene as an AHH inducer.

Poland and coworkers have extensively studied the induction of AHH and other enzymes by 2,3,7,8-TCDD and a variety of its congeners (50). This structure-activity relationship has been examined using a variety of PCDD (50) and PCDF (50, 197) congeners.

The AHH induction bioasssay utilizes a rat hepatoma cell line which possesses low basal AHH activity and is highly inducible for this enzyme. The amount of AHH induction is determined by the fluorimetric assay of 3-hydroxybenzo[a]pyrene formation from benzo[a]pyrene (the substrate for the AHH enzyme assay), or by the fluorimetric determination of 7-ethoxyresorufin O-deethylation. This technique has been used to examine individual halogenated aromatics, food, and environmental extracts (191). For example, several gelatin samples have been shown to contain pentachlorophenol and related PCDD impurities, including the 1,2,3,6,7,8- and 1,2,3,7,8,9-hexa- and 1,2,3,4,5,7,8-hepta-CDDs which exhibit AHH activity. The results confirmed the correlation between the concentration of the toxic dioxins in the samples and their bioassay 2,3,7,8-TCDD equivalents.

The binding of a specific ligand or mixture of ligands is determined by measuring the dose-dependent displacement of [^{3}H]-2,3,7,8-TCDD previously bound to the protein.

Toxicity to Cells Cultured in Vitro

One of the most characteristic responses to TCDD in vivo is hyperkeratosis in the skin. Recent work by Knutson and Poland has demonstrated that the hyperkeratinization response to TCDD exposure which occurs upon exposure to TCDD in vivo could be reproduced in an in vitro model system (192, 195).

When XB-cells derived from a mouse teratoma are cultured at high density (to prevent spontaneous diffentiation) along with lethally irradiated 3T3 cells, the addition of TCDD produces a dose-related keratinization response, as detected by red staining with Rhodanile blue. This is a direct effect of TCDD on XB cells, and will occur in the absence of the feeder cells if the teratoma cells are cultured in 3T3-conditioned media. XB cells contain the cytosol receptor, and respond to TCDD with a dose-related induction of AHH activity.

Other halogenated aromatic hydrocarbon congeners, including PCDDs, PCDFs, PCBPs, and azobenzenes, also induced keratinization although to a lesser extent than 2,3,7,8-TCDD. Keratinization could also be induced by some nonhalogenated aromatic hydrocarbons, such as benzanthracene and 5,6-benzoflavone (192). Since 2,3,7,8-TCDD is the most potent, but not the only inducer of this hyperkeratinization response, this effect is referred to as "dioxin-like" activity (196). Unrelated toxins were studied to determine if the keratinization response was due to nonspecific cell damage (192). The direct acting alkylating agents N-acetoxy-2-acetylaminofluorene and N-methyl-N'-nitrosoguanidine; inhibitors of nucleic acid synthesis, actinomycin D, bleomycin, and cordycepin; the spindle microtubule inhibitor, colchicine; kepone, a chlorinated hydrocarbon producing a toxic syndrome distinct from that of TCDD; and 2,6-dichlorobenzonitrile, an acnegen considered to have a different mechanism than TCDD all failed to produce keratinization in the XB/3T3 culture system (192). Furthermore, a correlation has been demonstrated between the degree of toxicity of various TCDD isomers and congeners and the extent of hyperkeratinization response (124, 192).

The XB/3T3 system is extremely sensitive, giving an induction of keratinization with as little as 3.2 picograms of TCDD (192). In addition, this system is particularly relevant as a model, since it deals with an induced differentiation of the intact cell using an endpoint (hyperkeratinization) known to be relevant to human exposure (i.e., chloracne) (196).

Section 8

RESEARCH NEEDS

INTRODUCTION

This section outlines research recommendations directly related to the potential formation of PCDFs, PCDDs, and other polychlorinated aromatics from askarel fluids. These recommendations, based on the findings presented in previous sections, reflect the identified information gaps regarding the generation of these compounds, their toxicity, methods of analysis, environmental pathways, and procedures for their destruction/removal.

GENERATION

PCDFs, PCDDs, PCBPs, and other polychlorinated aromatic compounds have been generated in association with PCB equipment fires and explosions. The quantities generated were found to vary significantly, depending on the type of equipment and reaction conditions. The probability of generation under normal operating conditions and in noncatastrophic malfunctions is still highly uncertain.

Since available information on both conditions and quantities of formation is insufficient and often contradictory, future studies should focus on better delineating the generation of PCDFs, PCDDs, and PCBPs. In parallel, standard and non-standard operating conditions should be compared with optimum formation conditions to determine (1) if formation is indeed possible, and (2) if the necessary precursors exist in PCB fluids. Operating conditions of primary interest include the following:

- Long-term high temperature overload in the presence of an oxygen source (e.g., water, O_2).
- Short-term catastrophic failure caused by internal arcing.
- Long-term partial discharge.
- Arcing in circuit breakers containing PCB-contaminated oil.

By conducting the above tests under controlled conditions, performing the analyses using the most advanced analytical techniques, and comparing the resulting data to

established theories, the above experiments would provide specific insights into the following:

- Reaction conditions favoring the formation of PCDFs from PCBs.
- Mechanisms and optimum reaction conditions for the formation of PCDDs from chlorophenols and chlorobenzenes.
- Chemistry and reaction conditions for the formation of PCBPs.
- Effects of dilution of PCBs in mineral oil on the formation of PCDDs, PCDFs, and other chlorinated species.

TOXICITY

Environmental contaminants become a concern when they affect human safety and the quality of the environment. It is therefore important to assess the risks resulting from exposure to the above compounds using the appropriate toxicological criteria. While other PCDD homologues have been studied to a lesser degree, the data on structure-biological activity relationships for 2,3,7,8-TCDD in laboratory animals are extensive. These data pertain to lethality, carcinogenicity, and reproductive effects.

Toxicological date for PCDFs and PCBPs are very limited, as are the data on the synergistic effects of two or more of the above by-products. For these compounds, specific data are lacking on acute lethality, chronic toxicity, carcinogenicity, teratogenic and reproductive effects, mutogenicity, and immunotoxicity. There are significant gaps in the relationship between the receptor theory and the induction of chloracne. Data on such effects as the extent of enzyme induction through specific receptor binding and cell keretinization should be acquired through bioassay techniques.

In particular, research should be focused on the human target of PCDDs and PCDFs in at least three areas:

- Dosage/time/response relationship for each major health effect actually seen in humans as a function of exposure to potential PCDDs and PCDFs.
- Mechanisms of action at the tissue level for each major health effect of these chemicals in humans.
- Quantitative risk assessments for each real, chronic health effect in humans under exposure conditions actually met in occupational or environmental situations.

CHEMICAL AND BIOASSAY ANALYSES

It is apparent that there are several areas of research which require additional emphasis in order to further advance the capabilities for analysis of PCDDs/PCDFs. Such areas include the following:

- Synthesis and isolation of additional pure PCDD/PCDF isomer standards for use in calibration and methods development.
- Synthesis of additional compounds which are chemically similar to PCDDs/ PCDFs, and which may represent interferences in PCDD/PCDF analysis (diphenylethers, hydroxylated- and methoxy-DPEs and PCBs) for methodology evaluation.
- Development of higher resolution GC and/or LC columns for isolation of PCDD/PCDF isomers.
- Research on more specific detection, quantification, and confirmation methods, including possibly positive and negative ion chemical ionization MS, atmospheric pressure ionization MS, and MS/MS.
- Research into an isomer-specific analytical method to differentiate between toxic and nontoxic isomers.
- Research into rapid chemical and/or biological screening methods.
- Standardization of sampling methods, particularly for such techniques as wipe tests, and establishment of criteria to identify the significance of analytical results obtained via different sampling methods (e.g., how the results of a wipe test relate to toxicological data).

ENVIRONMENTAL PERSISTENCE

The environmental pathways of these compounds need to be better defined. Neither the pathways nor the rates (other than general overall rates) for their physical migration and decay have been determined with any certainty. Photolysis, for example, has been called the main route of environmental degradation. However, this process requires a hydrogen donor, which is not likely to be present under most normal environmental conditions. Thus, further research is needed into photolysis to define the role and abundance of hydrogen donors, and the exact conditions under which measurable photolysis will occur in air, water, and soil.

In aquatic systems, photolysis would be limited because of the attenuation of light with depth; in soils, photolysis would be rapid on the surface. A longer half-life is therefore expected as depth increases.

The persistence of these compounds in both water and soil may also be controlled by their uptake/metabolism by biota, but the degree to which this occurs cannot be determined, based upon existing metabolic degradation research.

DESTRUCTION/REMOVAL

Since these compounds are both persistent in the environment and toxic to animals and humans, more research needs to be directed toward the development of chemical, physical, and/or biological methods for effectively treating wastes containing PCDFs, PCDDs, and PCBPs, as well as soil and water contaminated with these by-products.

Section 9

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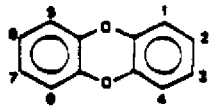
Appendix A

POLYCHLORINATED* DIBENZO-p-DIOXIN CONGENERS BY HOMOLOGUE AND ISOMER (NUMBER OF ISOMERS)**



Numbering system for congeners

MONOCHLORO-(2)

1- 2-

DICHLORO-(10)

1,2- 1,3- 1,4-
1,6- 1,7- 1,8-
1,9- 2,3- 2,7-
2,8-

TRICHLORO-(14)

1,2,3- 1,2,4-
1,2,6- 1,2,7-
1,2,8- 1,2,9-
1,3,6- 1,3,7-
1,3,8- 1,3,9-
1,4,6- 1,4,7-
1,7,8- 2,3,7-

TETRACHLORO-(22)

1,2,3,4- 1,2,3,6-
1,2,3,7- 1,2,3,8-
1,2,3,9- 1,2,4,6-
1,2,4,7- 1,2,4,8-
1,2,4,9- 1,2,6,8-
1,2,6,7- 1,2,7,8-
1,2,6,9- 1,2,8,9-
1,2,7,9- 1,3,6,9-
1,3,6,8- 1,3,7,9-
1,3,7,8- 1,4,7,8-
1,4,6,9- 2,3,7,8-

PENTACHLORO-(14)

1,2,3,4,6- 1,2,3,4,7-
1,2,3,6,7- 1,2,3,6,8-
1,2,3,6,9- 1,2,3,7,8-
1,2,4,7,9- 1,2,4,8,9-
1,2,4,6,7- 1,2,4,6,8-
1,2,4,6,9- 1,2,4,7,8-
1,2,4,7,9- 1,2,4,8,9-

HEXACHLORO-(10)

1,2,3,4,6,7-
1,2,3,4,6,8-
1,2,3,4,6,9-
1,2,3,4,7,8-
1,2,3,6,7,8-
1,2,3,6,7,9-
1,2,3,6,8,9-
1,2,3,7,8,9-
1,2,4,6,7,9-
1,2,4,6,8,9-

HEPTACHLORO-(2)

1,2,3,4,6,7,8-
1,2,3,4,6,7,9-

OCTACHLORO-(1)

1,2,3,4,6,7,8,9-

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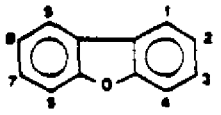
* EPA includes monochlorinated compounds under the general term, "polychlorinated."

** "Congener" is a general term used to mean any isomer of any homologue; thus, there are a total of 75 PCDD congeners. In the present document, "homologue" is defined as a group of isomers having a specific number of chlorine atoms; thus, the tetrachloro- homologue has 22 isomers. An "isomer" is defined by the numerical arrangement of chlorine atoms within the homologue.

POLYCHLORINATED DIBENZOFURAN CONGENERS BY HOMOLOGUE AND ISOMER- (NUMBER OF ISOMERS)



Numbering system for congeners

MONOCHLORO-(4)
1- 2- 3- 4-

DICHLORO-(16)
1,2- 1,3- 1,4-
2,3- 2,4- 3,4-
1,6- 1,7- 1,8-
1,9- 2,6- 2,7-
2,8- 3,6- 3,7-
4,6-

TRICHLORO-(28)
1,2,3- 1,2,4-
1,3,4- 2,3,4-
1,2,6- 1,2,7-
1,2,8- 1,2,9-
1,3,6- 1,3,7-
1,3,8- 1,3,9-
1,4,6- 1,4,7-
1,4,8- 1,4,9-
2,3,6- 2,3,7-
2,3,8- 1,7,8-
2,4,6- 2,4,7-
2,4,8- 1,6,8-
3,4,6- 3,4,7-
2,6,7- 1,6,7

TETRACHLORO-(38)
1,2,3,4- 1,2,3,6-
1,2,3,7- 1,2,3,8-
1,2,3,9- 1,2,4,6-
1,2,4,7- 1,2,4,8-
1,2,4,9- 1,3,4,6-
1,3,4,7- 1,3,4,8-
1,3,4,9- 2,3,4,6-
2,3,4,7- 2,3,4,8-
1,6,7,8- 1,2,6,7-
1,2,6,8- 1,2,6,9-
1,2,7,8- 1,2,7,9-
1,2,8,9- 1,3,6,7-
1,3,6,8- 1,3,6,9-
1,3,7,8- 1,3,7,9-
1,4,6,7- 1,4,6,8-
1,4,6,9- 1,4,7,8-
2,4,6,7- 2,4,6,8-
3,4,6,7- 2,3,6,7-
2,3,6,8- 2,3,7,8-

PENTACHLORO-(28)
1,2,3,4,6- 1,2,3,4,7-
1,2,3,4,8- 1,2,3,4,9-
1,2,3,6,7- 1,2,3,6,8-
1,2,3,6,9- 1,2,3,7,8-
1,2,3,7,9- 1,2,3,8,9-
1,2,3,6,7- 1,2,3,6,8-
1,2,4,6,9- 1,2,4,7,8-
1,2,4,7,9- 1,2,4,8,9-
1,2,4,6,7- 1,2,4,6,8-
1,3,4,6,9- 1,3,4,7,8-
1,3,4,7,9- 1,3,4,8,9-
2,3,4,6,7- 2,3,4,6,8-
1,4,6,7,8- 2,3,4,7,8-
1,3,6,7,8- 1,2,6,7,8-

HEXACHLORO-(16)
1,2,3,4,6,9- 1,2,3,4,6,8-
1,2,3,4,6,7- 1,2,3,4,7,8-
1,2,3,4,7,8,9- 1,2,3,6,7,8-
1,2,3,7,8,9- 1,2,3,6,7,9-
1,2,3,6,8,9- 1,2,4,6,7,8-
1,2,4,6,7,9- 1,2,4,6,8,9-
1,3,4,6,7,8- 1,3,4,6,7,9-
2,3,4,6,7,8- 1,2,3,4,7,9-

HEPTACHLORO-(4)
1,2,3,4,6,7,8-
1,2,3,4,6,7,9-
1,2,3,4,6,8,9-
1,2,3,4,7,8,9-

OCTACHLORO-(1)
1,2,3,4,6,7,8,9-

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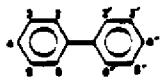
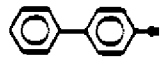
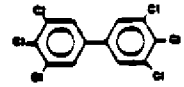
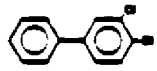
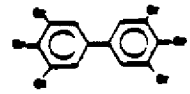
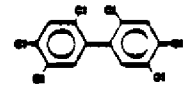
Appendix B

POTENCY OF HALOGENATED BIPHENYLS IN INDUCING HEPATIC ARYL HYDROCARBON HYDROXYLASE ACTIVITY IN CHICKEN EMBRYOS

Chicken embryos at 17 days of gestation were administered halogenated biphenyl compounds dissolved in 25 ul of p-dioxane. Twenty-four hours later, they were killed, and their livers were assayed for aryl hydrocarbon hydroxylase activity.

		ED_{50} (mol/egg)			ED_{50} (mol/egg)
1		inactive	9		4.4×10^{-9}
2		inactive	10		4.7×10^{-9}
3		inactive	11		3.4×10^{-9}
4		inactive	12		inactive
5		inactive	13		inactive
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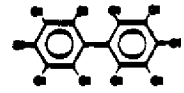
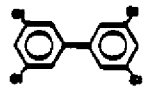
7		inactive	15		inactive
8		inactive	16		inactive

Appendix C

TRANSFORMER DATA FOR TESTS CONDUCTED BY CHITTIM, ET AL. (75)

Sample	Installation	Manufacturer*	Dielectric Strength (kV)	Power (kVA)	Volume (liters)	kVA/l
Group 1						
1-1	1957	FP	36	1,500	1,888	0.79
1-2	1961	FP	36	1,500	2,093	0.72
1-3	1963	FP	36	1,500	1,251	1.19
1-4	1963	FP	36	1,500	1,251	1.19
1-5	1963	FP	36	1,500	1,274	1.17
1-6	1965	FP	29	1,500	1,151	1.30
1-7	1970	FP	30	1,500	1,765	0.85
1-8	1970	FP	22	1,500	1,490	1.01
1-9	1971	FP	28	1,500	1,490	1.01
1-10	1974	FP	28	1,500	1,490	1.01
1-11	NEW	FP	-	1,500	-	-
Group 2						
2-1	1967	CGE	33	500	725	0.69
2-2	1967	CGE	31	1,000	938	1.07
2-3	1967	CGE	36	1,000	770	1.30
2-4	1967	CGE	36	1,866	1,268	1.47
2-5	1965	FP	36	700	660	1.06
2-6	1965	FP	36	1,500	1,251	1.20
2-7	1965	FP	36	2,500	2,084	1.20
2-8	1965	FP	36	2,500	1,092	2.29
Group 3						
3-1	1967	CGE	33	500	725	0.69
3-2	1967	CGE	41	1,000	770	1.30
3-3	1967	W	41	750	883	0.85
3-4	1967	W	41	1,200	1,087	1.10
3-5	1967	FP	31	750	829	0.90
3-6	1967	FP	36	1,000	838	1.19
Group 4						
4-1	1947	CGE	-	56	660	0.08
4-2	1947	CGE	-	10,000	6,900	1.45
4-3	1965	AD	-	-	-	-
4-4	1973	WA	-	-	-	-
4-5	NEW	CGE	-	-	-	-
4-6	NEW	W	-	-	-	-
4-7	NEW	FP	-	-	-	-

* FP - Ferranti-Packard
CGE - Canadian General Electric
W - Westinghouse
AD - Brown Boveri
WA - Foster Electric

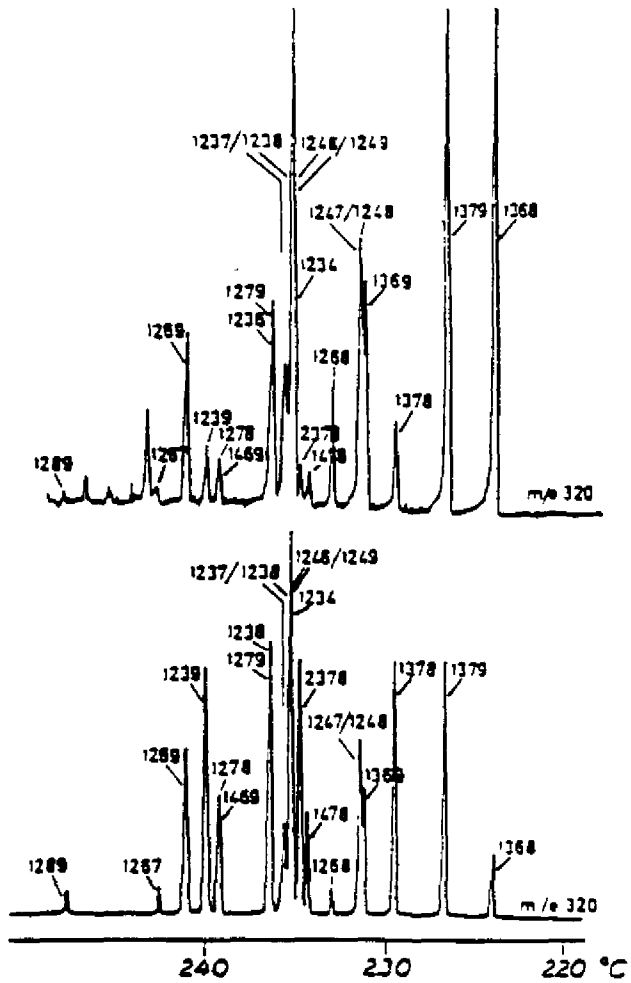


Figure C-1. Fragmentogram (Based on Mass Specific Detection) of a Fly Ash Sample (Above) and Standard Mixture of TCDD (Bottom) on a 55-m Silar 10-C Column (i.d. 0.26 mm) (113)

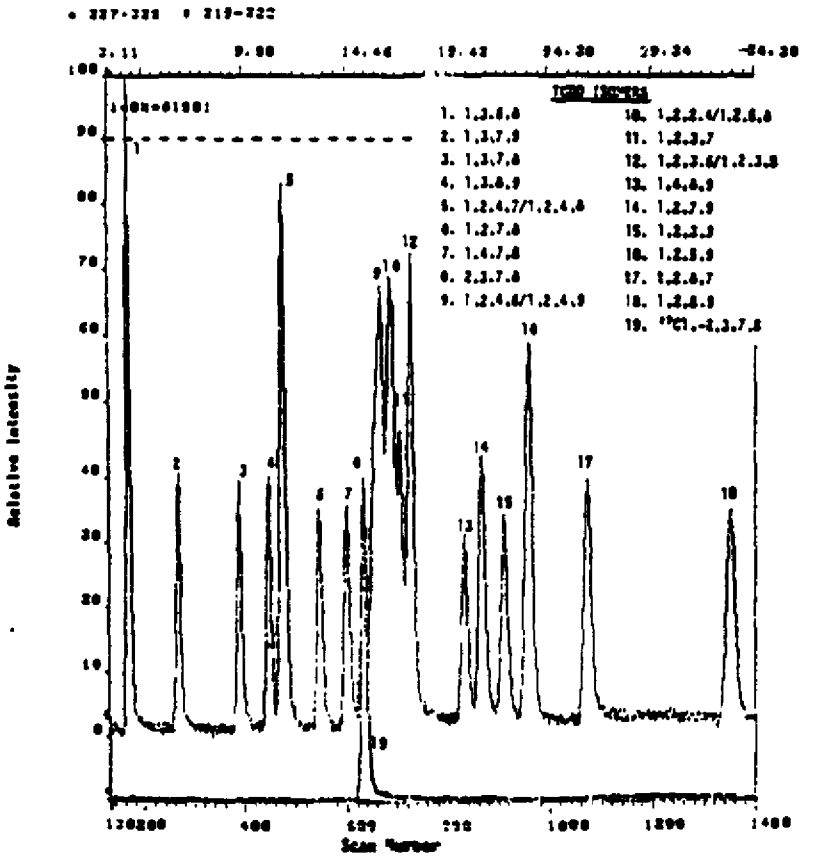


Figure C-2. Multiple Selected Ion Mass Chromatogram Obtained for Calibration Mixture Containing the 22 TCDD Isomers (Column: 50-m Hybrid Phase WCOT Glass Capillary) (Brehm Laboratory, Personal Communications)

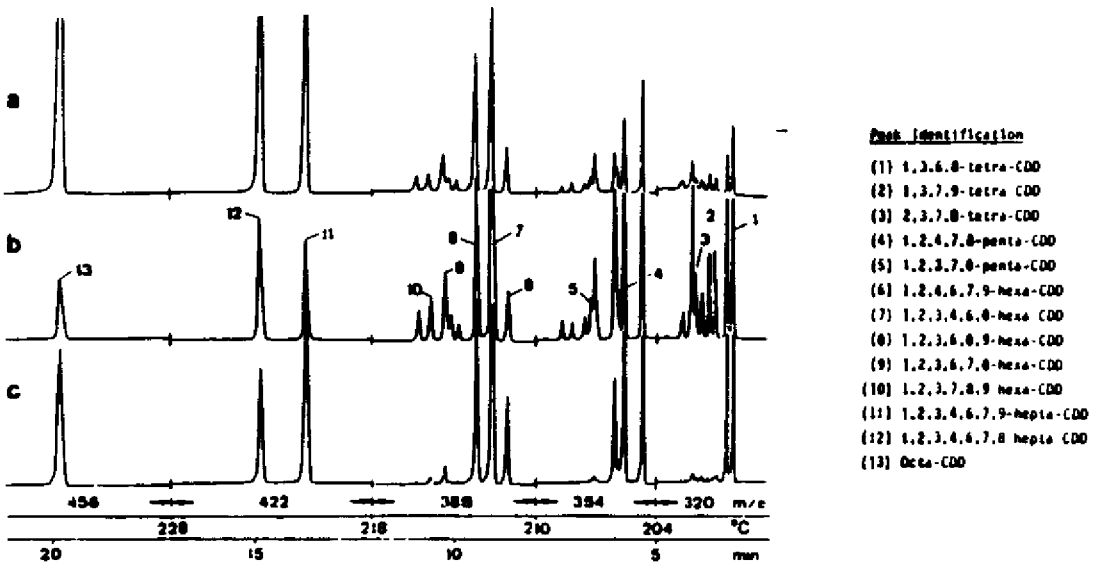


Figure C-3. Mass Fragmentograms (OV-17 Glass Capillary Column, 200-240°C) Showing Elution of PCDDs in (a) Fly Ash of a Municipal Incinerator, (b) Fly Ash of an Industrial Heating Facility, and (c) Micropyrolysis Sample of Combined 2,4,6-tri-, 2,3,4,6-tetra, and pentachlorophenate; m/e 320, 354, 388, 422, and 456 for tetra- to octa-CDDs (34)

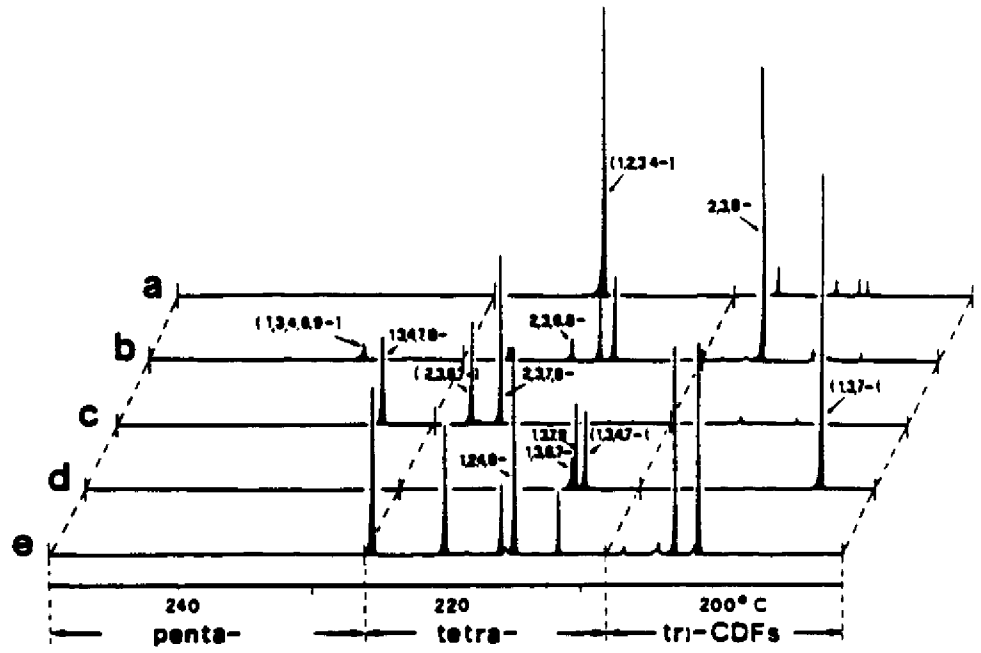


Figure C-4. Mass Fragmentograms (m/e 270, 304, and 338) of Pyrolyzed Pentachlorobiphenyls Showing Elution of tri-, tetra-, and penta-CDFs from (a) 2,3,4,5,6-, (b) 2,4,5,2',5'-, (c) 2,4,5,3',4'-, (d) 2,4,6,2',4'-, and (e) 2,3,6,2',5'-pentachlorobiphenyl (50-m OV-17 Glass Capillary Column, 100-160° at 20°/min, 160-250° at 2°C/min) (34)

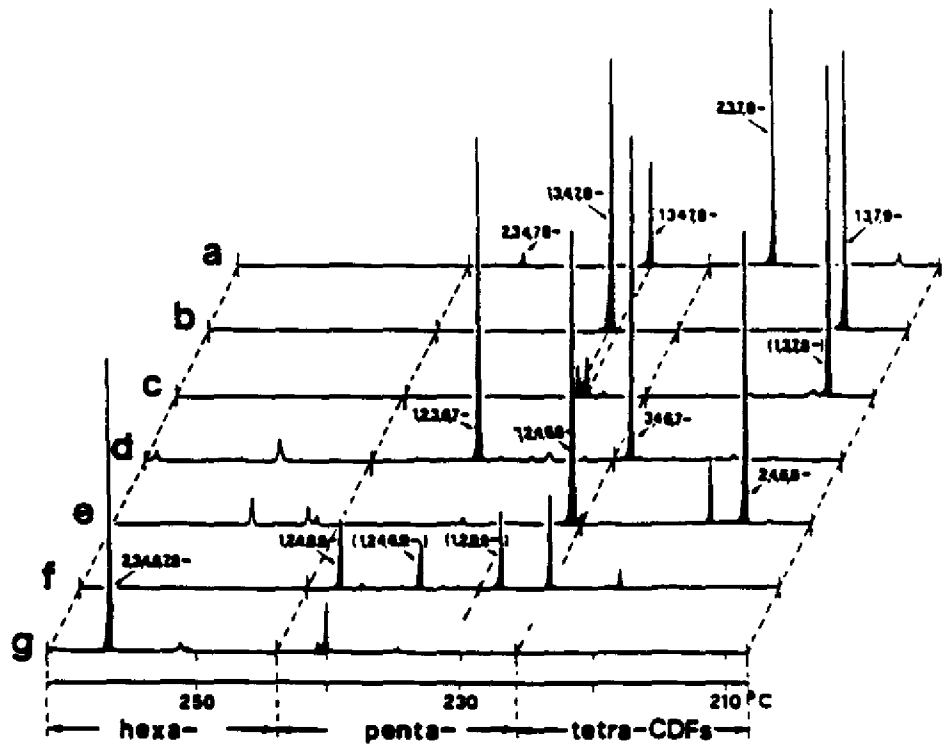


Figure C-5. Mass Fragmentograms (m/e 304, 338, and 372) of Pyrolyzed Hexachlorobiphenyls Showing Elution of tetra-, penta-, and hexa-CDFs from (a) 2,4,5,2',4',5'-, (b) 2,4,6,2',4',6'-, (c) 2,4,5,2',4',6'-, (d) 2,3,4,2',3',4'-, (e) 2,3,5,2',3',5', (f) 2,3,6,2',3',6'-, and (g) 3,4,5,3',4',5'-hexachlorobiphenyl (50-m OV-17 Glass Capillary Column, 100-160° at 20°/min, 160-250° at 2°C/min) (34)

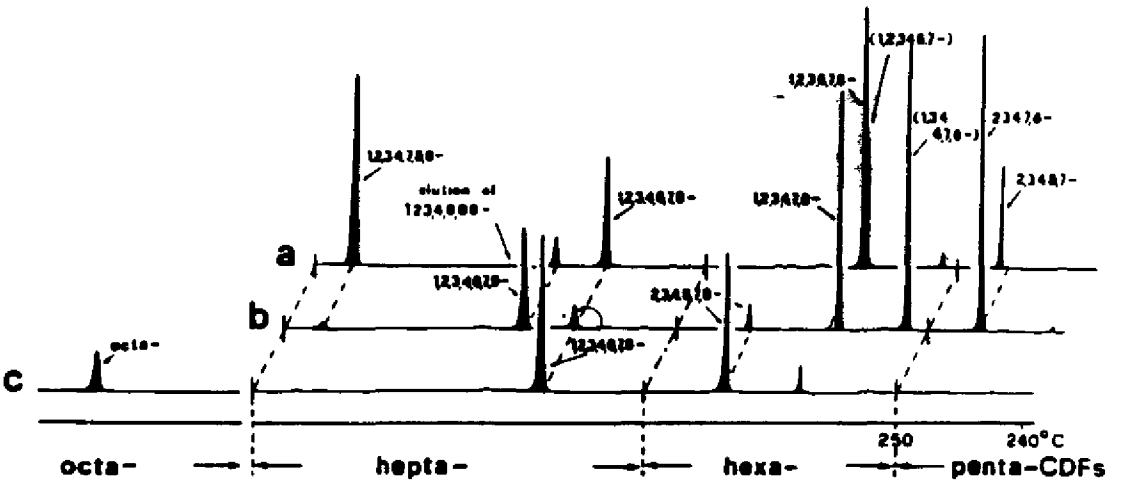


Figure C-6. Mass Fragmentograms (m/e 338, 372, 406, and 440) of Pyrolyzed Hepta- and Octachlorobiphenyls Showing Elution of penta-, hexa-, hepta-, and octa-CDF from (a) 2,3,4,5,2',3',4'-, (b) 2,3,4,5,2',4',5'-hepta, and (c) 2,3,4,5,2',3',4',5'-octachlorobiphenyl (50-m DV-17 Glass Capillary Column, 100-160° at 20°/min, 160-250° at 2°C/min) (34).

Appendix D

EXTRACTION METHODS FOR PCDDs AND PCDFs

U.S. Environmental Protection Agency (EPA) Procedures (153)

Acid-Extraction Procedure - Applied to Fish, Adipose Tissue, Milk, Water, Soil, and Sediment--

- Spike 10 to 20 g of sample with $^{37}Cl_4$-2,3,7,8-TCDD standard.
- Reflux with KOH.
- Extract with hexane.
- Remove and extract hexane fraction with concentrated H_2SO_4.
- Dry on sodium carbonate.
- Chromatograph on neutral alumina column using CCl_4 and CH_2Cl_2 to elute.

Neutral Extraction Procedure - Applied to Fish--

- Blend 15 to 20 g of sample with sodium sulfate and dry ice.
- Spike resultant powder with $^{37}Cl_4$-2,3,7,8-TCDD standard.
- Extract with acetonitrile.
- Remove acetonitrile fraction and extract with acetonitrile-saturated hexane.
- Discard hexane.
- Concentrate acetonitrile and replace with hexane.
- Chromatograph on florisil column, then on neutral alumina column, eluting sequentially with 100 percent hexane, 10 percent CH_2Cl_2-in-hexane, and 25 percent CH_2Cl_2-in-hexane.

U.S. Food and Drug Administration (FDA) Procedure (176) - Applied to Chemical Formulations and Fish

- Dissolve 20 g of sample in alkaline solution by agitating at room temperature for 2 to 3 hours.
- Extract with hexane.
- Remove hexane and extract with concentrated acid.
- Dry on sodium carbonate.
- Chromatograph on neutral alumina, eluting with 20 percent CCl_4-in-hexane, then CH_2Cl_2.
- HPLC on Zorbax-ODS, 40°C, methanol eluant.

U.S. Fish and Wildlife Service Procedure (177)

- Blend sample with sodium sulfate to free-flowing powder.
- Pick powder into glass column and extract with CH_2Cl_2 (200 ml for each g of fish).
- Evaporate solution to 50 ml.
- Add methanol and benzene to yield solution containing 20 percent CH_3OH and 5 percent benzene.
- Pass extract through column containing segments of potassium silicate and silica gel.
- Pass solution through carbon-glass fiber sorbent, and wash sorbent with $CH_2Cl_2/CH_3OH/C_6H_6$ (75/20/5).
- Elute with toluene.
- Chromatograph on column containing H_2SO_4 dispersed on silica gel and cesium.
- Chromatograph on acid alumina, eluting with hexane and CH_2Cl_2-hexane.

Dow Chemical Company Procedures

Acid Extraction Procedure (178, 179) - Applied to Fish and Milk--

- Spike 10 to 20 g of sample with ^{13}C-TCDD internal standard (and/or other PCDD standards).
- Shake with concentrated HCl for 1 hour.
- Extract with hexane (overnight shaking plus 3 additional hours).
- Pass hexane extract through combined column of silica, concentrated H_2SO_4 on silica, and 1M KOH on silica (i.e., 22 percent H_2SO_4, 44 percent H_2SO_4 on silica columns, and NaOH on silica columns are used alternatively).
- Pass hexane extract through second dual column of silver nitrate on silica and basic alumina.
- Clean up dioxin fractions using normal phase silica (Zorbax-SIL) HPLC, then reverse phase HPLC (Zorbax-ODS, methanol solvent).

Neutral Extraction Procedure (180) - Applied to Particulates (Fly Ash, Dust, Urban Particulates), Municipal Sludge, and Soils--

- Spike 50 mg to 100 g of sample with ^{13}C-TCDD, ^{13}C-HCDD, and ^{13}C-OCDD internal standards.
- Soxhlet extract sample with benzene for at least 16 hours.
- Concentrate extract using Snyder column.

- Subject to liquid chromatographic and HPLC cleanup similar to that described for acid extraction procedure.

Wright State University-Brehm Laboratory Procedures*

Procedure for Soils, Chemical Wastes, and Aqueous Effluents (180, 181)--

Soils

- Spike 1 to 10 g of soil with ^{37}Cl-TCDD internal standard (and/or other internal standards).
- Extract spiked soil in bottle with petroleum ether or hexane-methanol by shaking for period of 20 minutes to overnight.
- Alternatively, extract spiked soil in a Soxhlet apparatus with methylene chloride or benzene, followed by concentration of extract on a Snyder column.
- Wash the organic phase successively with 20 percent KOH, water, concentrated H_2SO_4, and water, discarding the wash solutions.
- Dry organic extract over sodium sulfate.
- Pass extract through basic alumina column.
- Clean up dioxin fractions, using normal phase silica (Zorbax-SIL) and reverse phase (Zorbax-ODS) HPLC.

Chemical Wastes

- Spike 1 to 10 g of sample with ^{37}Cl-TCDD internal standard (and/or other labelled standards).
- Extract spiked sample in bottle with methanol and petroleum ether by shaking for period of 20 minutes to overnight.
- Continue with cleanup as in soils.

Aqueous Effluents

- Spike 10 to 20 g of sample with ^{37}Cl-TCDD internal standard (and/or other internal standards).
- Extract spiked sample in bottle with petroleum ether by shaking for period of 20 minutes.
- Continue with cleanup as in soils.

Procedure for Particulates and Other Samples from Flue Gas Stack Sampling Trains (160, 182)--

- Spike appropriate quantities of organic and aqueous liquids, particulates, XAD-2 resins, and other sorbents with ^{37}Cl-labelled and/or ^{13}C-labelled PCDDs/PCDFs.
- Extract liquid samples with petroleum ether by shaking in bottle for 1 hour.
- Extract solid samples in a Soxhlet apparatus with benzene.
- Wash extracts successively with KOH, water, and H_2SO_4, and discard washings.
- Pass extracts through a combination macrocolumn of silica, 33 percent NaOH-silica, 44 percent H_2SO_4-silica and silica, eluting with hexane.
- Pass eluate through a Woelm basic alumina minicolumn, eluting with 3 percent CH_2Cl_2-in-hexane and 50 percent CH_2Cl_2-in-hexane, collecting appropriate fractions.

Umea University (Sweden) Procedures

Procedure for PCB Cleanup (10)--

- Chromatograph 100 mg used PCB on an alumina microcolumn (1.0 g basic alumina, Woelm, in Pasteur pipette), eluting with 10 ml of n-hexane.
- Recover PCDFs by eluting with 6 ml of CH_2Cl_2 after careful evaporation to dryness.
- Chromatograph on a second alumina microcolumn, eluting with 10 ml of 4 percent CH_2Cl_2 in n-hexane.
- Recover PCDFs and other chlorinated tricylic aromatic compounds with 10 ml of 50 percent CH_2Cl_2 in n-hexane.

Procedure for Fly Ash, Soot, Wipe Tests (3, 183, 184)--

- Spike 1 g sample with 1 ppb of 2,3,7,8-$^{13}C_{12}$-tetra-CDD, 2,3,7,8-$^{37}Cl_4$-tetra-CDF, and $^{37}Cl_8$-octa-CDD.
- Shake for 1 hour with 10 ml of 1M HCl.
- Filter by suction using Buchner funnel, and wash with water.
- Dry in air, and extract the dried material with approximately 100 ml toluene in a Soxhlet extractor for 48 hours with a speed of 1 to 2 ml/min.
- Reduce toluene to 100 ul by evaporation on a rotavapor using some external heating.

- Eliminate remaining toluene by a purified N_2 flush without external heating.
- Dissolve the residue in 2 ml of n-hexane.
- Chromatograph on a silica gel column (0.5 g Kieselgel 60, 70 to 230 mesh (Merck)), eluting with 5 ml of n-hexane.
- Reduce n-hexane under N_2 to 2 ml by evaporation.
- Analyze for PCBPs.
- Separate PCDDs and PCDFs using an Alox column (1.0 basic Alox akt 1, Woelm, ICN West Germany).
- Discard the first fraction, i.e., 10 ml of n-hexane: CH_2Cl_2 (98:2).
- Collect the second fraction, i.e., 10 ml of n-hexane: CH_2Cl_2 (1:1), and evaporate to dryness under N_2.
- Dissolve the residue in 100 ul of solvent.

Procedure for Blood Sample (79, 135)--

- Add 10 ml of n-hexane to 10 ml of blood plasma.
- Spike with 0.1 ng of $^{13}C_{12}$-2,3,7,8-tetra-CDD.
- Add 10 ml of acetonitrile saturated with n-hexane (three times).
- Shake mixture and centrifuge.
- Treat the combined acetonitrile phases with 10 ml of 1 percent aqueous sodium sulfate solution.
- Extract three times with 20 ml of n-hexane.
- Dry the combined n-hexane layers with sodium sulfate, and concentrate to 2 ml.
- Chromatograph on alumina column (1 g alumina).
- Elute first with 10 ml of 2 percent CH_2Cl_2 in n-hexane, and discard the eluate; then elute with 10 ml of 50 percent CH_2Cl_2 in n-hexane.
- Concentrate the eluate.

Appendix E

GLOSSARY AND ABBREVIATIONS

AHH	Aryl hydrocarbon hydroxylase; an enzyme.
ALA	δ -aminolevulinic acid.
Aroclor	Monsanto trademark for PCBs.
Askarel	A generic descriptive name for synthetic dielectric material; for the purpose of this report, usually a mixture of PCBs and chlorinated benzenes.
Chloracne	An acne-like dermatological disorder due to the exposure to chlorine compounds.
Congener	A general term used to mean any isomer of any homologue.
Dioxin	Dibenzo-p-dioxin, a triple-ring organic compound consisting of two benze rings connected by a third ring through a pair of oxygen atoms; in popular parlance, dioxin is used for the 2,3,7,8-tetrachloro isomer.
Edema	An abnormal accumulation of fluid in the body.
Fragmentogram	The term used for the output from a mass specific detector mass spectrometer.
Furan	Specifically, a five-sided aromatic hydrocarbon with four carbon atoms and one oxygen atom in the ring; in general, the term is used for the dibenzofuran and its chlorinated isomers.
Homologue	A group of isomers having a specific number of chlorine atoms; thus, the 22 tetrachloro isomers would be the tetrachloro-homologue.
In vitro	Outside the living body and in an artificial environment.
In vivo	In the living body of a plant or animal.
Isomer	One of two or more molecules having the same number and kind of atoms, but differing in respect to the arrangement or configuration of the atoms.
Kanechlor	Japanese trademark for PCBs.
PCB	Polychlorinated biphenyl.
PCBP	Polychlorinated biphenylene.

PCCY	Polychlorinated chrysene.
PCDD	Polychlorinated dibenzo-p-dioxin; Dioxin.
PCDF	Polychlorinated dibenzofuran; Furan.
PCDPE	Polychlorinated diphenyl ether.
PCPY	Polychlorinated pyrene.
PCQ	Polychlorinated quarterphenyl.
PCQE	Polychlorinated quarterphenyl ethers.
PCT	Polychlorinated terphenyl.
PCX	Polychlorinated xanthene.
Photolysis	Decomposition of a compound into simpler units as a result of absorbing radiation.
Polychlorinated	For the purposes of this report, substituted with one or more chlorine atoms.
Precursor	An intermediate compound in the synthesis of a specific substance; a substance from which another substance is formed.
Predioxin	A diphenyl ether compound with a chlorine atom and hydroxyl group positioned such that they can react and form an oxygen bond between the two rings.
TCDD	Tetrachlorodibenzo-p-dioxin.
TCP	Trichlorophenol.
UV	Ultraviolet radiation.
Wipe Test	A sampling method where a specific area is wiped down and the wiping materials analyzed.
Yusho	Rice oil disease.

Appendix F

MATRIX OF REFERENCES FOR PCBs

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-KINS	FUR-ANS	OTH HCs	CH/H CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	HUM TOX	QUIN TOX	ACCD INCD	ENV IMPT	TOHS INFO	TRT/ DISP
ABATE L .ET AL MORTALITY AND BIRTH DEFECTS FROM 1976 TO 1979 IN THE POPULATION LIVING IN THE TCDD POLLUTED AREA OF SEVESO IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER, ET AL .EDS PERGAMON PRESS.OLNOPORO. N Y .1982 PP 871-897		X											X		X			
ACKERMAN.D G .ET AL DESTROYING CHEMICAL WASTES IN COMMERCIAL SCALE INCINERATORS FACILITY REPORT NO 6. ROLLINS ENVIRONMENTAL SERVICE, DEER PARK, TEXAS TRW DEFENSE AND SPACE SYSTEMS GROUP.REDONDO BEACH,CALIF . 1977.	X						X										X	X
ACKERMAN.D G..ET AL GUIDELINES FOR THE DISPOSAL OF PCBS AND PCB ITEMS BY INCANAL OBSTRUCTION EPA-600/2-81-022 .TRW.REDONDO BEACH,CALIF .FEBRUARY 1981	X						X											X
ACUREX CORPORATION DISPOSAL OF POLYCHLORINATED BIPHENYLS (PCBS) AND PCB-CONTAMINATED MATERIALS. VOL. 4: TSSI INCINERATION OF ELECTRICAL CAPACITORS CONTAINING PCBS EPOI FP-1287. ELECTRIC POWER RESEARCH INSTITUTE. PALO ALTO.CALIF .SEPTEMBER 1989.	X						X										X	X
ADAMOLI.P .ET AL ANALYSIS OF 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN IN THE SEVESO AREA IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL.ED ECOL BULL (STOCKHOLM),27: 31-38.1978		X									X				X			

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIB-XINS	FUR-ANS	OTH HCs	CHEM CHAR	D/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD THCB	ENV IMPT	TRNS INFO	TXT/ DISP
AHLING,B DESTRUCTION OF CHLORINATED HYDROCARBONS IN A CEMENT KILN ENVIRON SCI.TECHNOL .13:1377-1379. 1979	X						X											X
AHLING,B .AND A LINDSKOG EMISSION OF CHLORINATED ORGANIC SUBSTANCES FROM COMBUSTION IN: CHLORINATED DIBENZODIOXINS AND RELATED COMPOUNDS.IMPACT ON THE ENVIRONMENT. O. HUTZINGER,ET AL., EDS PERGAMON PRESS.OXFORD,N.Y., 1982. PP.215-223.	X	X	X	X			X											
AHLING,B .AND A.LINDSKOG. THERMAL DESTRUCTION OF PCB AND HEXACHLOROBENZENE. SCI.TOTAL ENVIRON .10:21-29, 1978.	X						X											X
AHLING,B .ET AL. FORMATION OF POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS DURING COMBUSTION OF A 2,4,5-T FORMULATION. CHEMOSPHERE,6:461-468,1977.		X	X				X											
AKEHURST,B PHOTOCHEMICAL REACTIONS OF PHENOXY ACIDS AND DIOXINS. IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS. C.RAMEL,ED. ECOL BULL. (STOCKHOLM),27:75-81.1979.		X						X										
AKEHURST,B PHOTODECHLORINATION OF HALOAROMATIC COMPOUNDS. IN: DIOXIN, TOXICOLOGICAL AND CHEMICAL ASPECTS. F.CATTABENI,A.CAVALLARO,AND G GALLI, EDS SPECTRUM PUBLICATIONS,NEW YORK, 1979 PP.191-193.		X		X		X		X										

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-MIMS	FUR-ANS	STA NCs	CHEM CHAR	D/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	HFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	FANS INFO	TBT/ DISP
ALBRO,P W .AND B J CORBETT EXTRACTION AND CLEAN-UP OF ANIMAL TISSUES FOR SUBSEQUENT DETERMINATION OF MIXTURES OF CHLORINATED DIBENZO P-DIOXINE AND DIBENZOFURANS. ENVIRONMENT.6: 301-305.1977		X	X								X			X				
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ALLEN,J.R .AND D.H.NORDACK PATHOBIOLOGICAL RESPONSES OF PRIMATES TO POLYCHLORINATED BIPHENYL COMPOUNDS IN: CONFERENCE PROCEEDINGS, NATIONAL CONFERENCE ON POLYCHLORINATED BIPHENYLS (NOVEMBER 1975, CHICAGO, ILLINOIS) EPA-560/6-75-004 MARCH 1976 PP 43-47.	X													X				
ALLEN J R ,ET AL MORPHOLOGICAL CHANGES IN MONKEYS CONSUMING A DIET CONTAINING LOW LEVELS OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN FOOD COSMET TOXICOL .15:401-410,1977		X												X				
AMERICAN CHEMICAL SOCIETY GUIDELINES FOR DATA ACQUISITION AND DATA QUALITY EVALUATION IN ENVIRONMENTAL CHEMISTRY ANAL.CHEM .52:2348-2349. 1980									X									

F-4

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-XENT	FUD-AMS	BIN HCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	RFC CHEM	ANAL CHEM	METB CHEM	MAM TOX	ANIM TOX	ACCD THCD	ENV IMPT	TRNS INFO	TRT/ DISP
AMERICAN MEDICAL ASSOCIATION. THE HEALTH EFFECTS OF "AGENT ORANGE" AND POLYCHLORINATED DIOXIN CONTAMINANTS. CHICAGO,OCTOBER 1981		x											x		x			
ANILINE,D PREPARATION OF CHLOROBENZO-P-DIOXINE FOR TOXICOLOGICAL EVALUATION IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR,ED. ADV CHEM SER.,120:126-135,1973.		x		x														
ARTHUR D LITTLE STUDY ON STATE-OF-THE-ART OF DIOXIN FROM COMBUSTION SOURCES AMERICAN SOCIETY OF MECHANICAL ENGINEERS,NEW YORK,1981.		x		x	x	x							x	x				
BAARD,A J ,ET AL INDUCTION OF RAT HEPATIC GLUTATHIONE-S-TRANSFERASE ACTIVITIES BY 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN:CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL.,EDS PERGAMON PRESS,ELMSFORD,N.Y ,1982 PP.393-401		x												x				
BAUCHMAN,R ,AND O MENZLOCH AN IMPROVED ANALYSIS FOR TETRACHLORODIBENZO-P-DIOXINE. IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR,ED ADV CHEM SER 120:92-104,1973		x									x							
BAUCHMAN,R D TCDD AND INDUSTRIAL ACCIDENTS PH D DISSERTATION,HARVARD UNIVERSITY,1974 EXCERPTED IN T UNGERSTOE THE PENDULUM AND THE TOXIC CLOUD YALE UNIVERSITY PRESS,NEW HAVEN,CONN ,1978 PP 148-158		x											x		x			

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-KING	FMS-ANS	BIO MCo	CHEM CHAR	D/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TOT/ DISP
BERRY,R I NEW WAYS TO DESTROY PCBS. CHEM ENG .90(16):37-41,1983	X										-							X
BIROS,F J ,A C WALKER AND A.MEDHEDOV. POLYCHLORINATED BIPHENYLS IN HUMAN ADIPOSE TISSUE. BULL ENVIRON.CONTAM. TOXIC ,9:317-323,1973	X										X		M					
BLAKED,W H ,ET AL. DETERMINATION OF CHLORINATED DIBENZO-P-DIOXINS IN PENTACHLOROPHENOL BY GAS CHROMATOGRAPHY MASS SPECTROMETRY ANAL CHEM.,48:884-909,1976		X									X							
BOLTON,L DEVELOP DIOXIN: MS SOLUTION IN DIONI. CHEM ENG.,84(22):78,1978.		X													X			
BOSTICK,W D ,ET AL. INDUCTION OF O-DEETHYLASE ACTIVITY AS AN INDEX TO EXPOSURE TO COAL-DERIVED PRODUCTS AND TRACE ENVIRONMENTAL POLLUTANTS CLIN CHEM ,27:1816-1823,1981	X		X									X						
BOTOE,C .A HENDLI,AND F.AL-HAIOUE ON THE DEGRADATION OF 2,3,7,8-TETRACHLORODIBENZO-PARADIOXIN (TCDD) BY MEANS OF A NEW CLASS OF CHLORODIOXIDES ENVIRON SCI TECHNOL ,13:229-231,1979		X			X													
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BOTTINI,A T.,AND J.D DOUGHTY. MECHANISMS FOR LIGHTD PHASE HYDROLYSES OF CHLOROBENZENE AND HALOTOLUENES J.AM CHEM SOC ,79:1490-1496.1957.			X		X													

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-XENO	FUR-ANS	BIN MCs	CHEM CHAR	D/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TOT/ DISP
BOWES,G W ET AL. GAS CHROMATOGRAPHIC CHARACTERISTICS OF AUTHENTIC CHLORINATED DIBENZOFURANS: IDENTIFICATION OF TWO ISOMERS IN AMERICAN AND JAPANESE POLYCHLORINATED BIPHENYLS J AGRIC.FOOD CHEM.,23(6):1205-1208,1975	X		X								X							
BOWES,G W ,ET AL. IDENTIFICATION OF CHLORINATED DIBENZOFURANS IN AMERICAN POLYCHLORINATED BIPHENYLS. NATURE, 256:380-387.1975	X		X								X							
BOWES,G W.,ET AL. ISOLATION AND IDENTIFICATION OF CHLORINATED DIBENZOFURANS FROM POLYCHLORINATED BIPHENYLS (PCB) AND FROM YUSHO RICE OIL CONTAINING PCB. IN: BIOMIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F.COTTA-RAMUSINO,A CAVALLARO,AND E GALLI,EDS SPECTRUM PUBLICATIONS,NEW YORK,1978 PP 89-99	X		X								X				X			
BRANDLAW,J ,AND J.L CASTENLINE INDUCTION OF ENZYME ACTIVITY IN CELL CULTURE: A RAPID SCREEN FOR DETECTION OF PLANAR POLYCHLORINATED ORGANIC COMPOUNDS J ASSOC.OFF ANAL.CHEM ,62: 904-916,1979		X	X									X		X				
BROADHURST,A E. USE AND REPLACEABILITY OF POLYCHLORINATED BIPHENYLS, ENVIRON.HEALTH PERSPECT.,EXPERIMENTAL ISSUE E:91-102.1976.	X																X	
BROWN,J.F.,JR HUMAN HEALTH EFFECTS OF ELECTRICAL GRADE PCB-E. GENERAL ELECTRIC RESEARCH AND DEVELOPMENT CENTER,SCHENECTADY,N Y ,1981	X		X		X							X	X	X	X			

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	DIB-HINS	FUR-ANS	DIN HCs	CHEM CHAR	D/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	IRMS INFO	TRI/ DISP
BRUCE,R NEW MACHINE TURNS PCB INTO SALT ARGUS (NEWSPAPER), JAN 13, 1982	X																	X
BRUNLEY,W C ,ET AL LOW-RESOLUTION MULTIPLE ION DETECTION GAS CHROMATOGRAPHIC-MASS SPECTROMETRIC COMPARISON OF SIX EXTRACTION-CLEANUP METHODS FOR DETERMINING 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN FISH J.AGRIC. FOOD CHEM ,28:1040-1046,1981		X							X		X							
BUHS R A ,ET AL TRACE CHEMISTRIES OF FIRE: A SOURCE OF CHLORINATED DIOXINS, SCIENCE,213(4507):305-310, OCT 24,1980		X					X											
BURGES E J ,JR SUMMARY: CONFERENCE ON DIBENZODIOXINS AND DIBENZOFURANS, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SERVICES, APRIL 2-3 1973, ENVIRON HEALTH PERSPEC ,(SPECIAL ISSUE 5):279-283,1973		X	X		X	X								X	X			
BUSER,H R ANALYSIS OF POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS IN CHLORINATED PHENOLS BY GAS FRAGMENTOGRAPHY J CHROMATOGR , 7:895-3(0,1979		X	X								X							
BUSER,H R ANALYSIS OF TCDD B BY GAS CHROMATOGRAPHY-MASS SPECTROMETRY USING GLASS CAPILLARY COLUMNS IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND C GALLI,EDS SPECTRUM PUBLICATIONS, NEW YORK,1978 PP 87-4]		X									X							

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	DIN HCs	CHEM CHAR	O/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/DISP
BUSER H R DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN ENVIRONMENTAL SAMPLES BY HIGH-RESOLUTION GAS CHROMATOGRAPHY AND LOW RESOLUTION MASS SPECTROMETRY ANAL CHEM., 49:918-920,1977		X									X							
BUSER,H R FORMATION AND IDENTIFICATION OF TETRA- AND PENTACHLORODIBENZO-P-DIOXINS FROM PHOTOLYSIS OF TWO ISOMERIC HEXACHLORODIBENZO-P-DIOXINS. CHEMOSPHERE,8:251-257,1979		X				X		X										
BUSER,H R FORMATION OF POLYCHLORINATED DIBENZOFURANS (PCDFS) AND DIBENZO-P-DIOXINS (PCDDS) FROM THE PYROLYSIS OF CHLOROBENZENES CHEMOSPHERE,8:415-424,1979.		X	X				X											
BUSER,H R. HIGH-RESOLUTION GAS CHROMATOGRAPHY OF THE 22 TETRACHLORODIBENZO-P-DIOXIN (TCDD) ISOMERS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O.HUTZINGER,ET AL.,EDS PERGAMON PRESS,ELMSFORD,N Y.,1982 PP 15-24		X									X							
BUSER,H R HIGH-RESOLUTION GAS CHROMATOGRAPHY OF POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS ANAL. CHEM.,48:1553-1557,1976.		X	X								X							
BUSER,H R POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS:FORMATION,OCCURRENCE AND ANALYSIS OF ENVIRONMENTALLY HAZARDOUS COMPOUNDS PH D THESIS UNIVERSITY OF UMEA UMEA, SWEDEN,1978		X	X		X						X							

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-MTHD	FUR-AND	OTH PCs	CHEM CHAR	D/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	MAN TOX	OMIN TOX	ACCD INCD	ENV IMPT	TOXC INFO	FDT/ DISP
DUDEK,M D. POLYCHLORINATED DIBENZO-P-DIOXINS: SEPARATION AND IDENTIFICATION OF ISOMERS BY GAS CHROMATOGRAPHY-MASS SPECTROMETRY J CHROMATOGR .114:99-106.1975.		X									X							
DUDEK,M D. PREPARATION OF ANALYTATIVE STANDARD MIXTURES OF POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS BY ULTRAVIOLET AND GAMMA IRRADIATION OF THE OCTACHLORO COMPOUNDS J.CHROMATOGR .103:303-307,1976		X	X							X	X							
DUDEK,M D ,AND C RAPPE FORMATION OF POLYCHLORINATED DIBENZOFURANS (PCDFS) FROM THE PYROLYSIS OF INDIVIDUAL PCB ISOMERS CHEMOSPHERE,8:157-174,1979	X		X			X	X											
DUDEK M A.,AND C.RAPPE HIGH-RESOLUTION GAS CHROMATOGRAPHY OF THE 22 TETRACHLORINATED DIBENZO-P-DIOXIN ISOMERS. ANAL CHEM .52:2057-2062.1980		X									X							
DUDEK,M.D.,AND C.RAPPE IDENTIFICATION OF CHLORINATION PATTERNS IN POLYCHLORINATED DIBENZO-P-DIOXINS (PCDDS) BY MASS SPECTROMETRY CHEMOSPHERE,8:199-811,1979		X									X							
DUDEK,M.M .AND M P GOODHART. DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN AT PARTS PER BILLION LEVELS IN TECHNICAL-GRADE 2,4,5-TRICHLOROPHENOXYACETIC ACID IN 2,4,5-I ALKYL ESTER AND 2,4,5-T AMINE SALT HERBICIDE FORMULATIONS BY GRADIENTPOLE MASS FRAGMENTOGRAPHY J.CHROMATOGR . 98:71-77,1974		X									X							

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-KINS	FUR-ANS	BIN MCs	CHEM CHAR	D/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	RFG CHEM	ANAL CHEM	METD CHEM	MAM TOX	AMIN TOX	ACCD INCD	ENV IMPT	TAND INFO	TOT/ DISP
BUSER,H R .AND H P BOSSHARDT DETERMINATION OF POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS IN COMMERCIAL PENTACHLOROPHENOLS BY CONDITIONED GAS CHROMATOGRAPHY-MASS SPECTROMETRY J ASSOC.OFF.ANAL.CHEM.,58:568-568,1976		X	X								X							
BUSER,H R .AND H.P BOSSHARDT POLYCHLORIERTE DIBENZO-P-DIOXINE, DIBENZOFURANE UND DERIVATE IN DER ASCHE KONTAMINIERTER UND INDUSTRIELLER VERBRENNUNGSANLAGE MITT.GEB LEBENSMITTELUNTERS.HYG .,68:1982-199,1978		X	X				X											
BUSER H R.,C RAPPE,AND A SAHA, POLYCHLORINATED DIBENZOFURANS (PCDFS) FOUND IN YUSHO OIL AND IN USED JAPANESE PCB. CHEMOSPHERE,7:439-448,1978	X		X		X													
BUSER,H R .ET AL IDENTIFICATION OF POLYCHLORINATED DIBENZOFURAN ISOMERS IN FLY ASH AND PCB PYROLYSES CHEMOSPHERE,7:419-428,1978	X		X				X											
BUSER,H R .H.-P BOSSHARDT,AND C RAPPE FORMATION OF POLYCHLORINATED DIBENZOFURANS (PCDFS) FROM THE PYROLYSIS OF PCBS. CHEMOSPHERE,7:109-118, 1978.	X		0				X											
BUSER,H R .H.-P BOSSHARDT,AND C RAPPE IDENTIFICATION OF POLYCHLORINATED DIBENZO-P-DIOXIN ISOMERS FOUND IN FLY ASH CHEMOSPHERE,7:160-172,1978.		X					X				X							
CANON] I .ET AL LABORATORY INVESTIGATION FOR THE MICROBIOLOGICAL DEGRADATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN SOIL BY ADDITION OF ORGANIC COMPOST IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,000 PERGAMON PRESS,OXFORD,N Y 1982 PP 99-103		X										X						

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REFERENCE	PCDs	BIO-KINS	FUR-ANS	OTH MCs	CMER CHAR	S/T CMER	TEMP CMER	PHOT CMER	ENV CMER	MFG CMER	ANAL CMER	METB CMER	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
CANTONI L ET AL INDUCTION OF MIXED FUNCTION OXIDASE BY CHRONIC TREATMENT WITH 2 3 7,8-TETRACHLORO-DIBENZO-P-DIOXIN IN FEMALE RATS TOXICOLOGY,37: 159-167 1981		X										X						
CARLSTEDT-DUKE J ,ET AL THE DETECTION AND FUNCTION OF THE CYTOSOLIC RECEPTOR FOR 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) AND RELATED CARCINOGENS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS,IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL ,EDS PERGAMON PRESS ELMSFORD,N Y ,1982 PP 255-265.		X												X				
CARRERI V REVIEW OF THE EVENTS WHICH OCCURRED IN SEVESO. IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND G.GALLI EDS SPECTRUM PUBLICATIONS,NEW YORK, 1978 PP 1-7		X													X			
CAVALLARO A ,ET AL A METHOD FOR THE DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN AT P P T. LEVELS IN VEGETABLES BY HIGH-RESOLUTION GAS CHROMATOGRAPHY AND LOW-RESOLUTION MASS SPECTROMETRY CHEMOSPHERE,8: 623-628,1980		X									X							
CAVALLARO,A ,ET AL NEGATIVE ION CHEMICAL IONIZATION MS AS A STRUCTURE TOOL IN THE DETERMINATION OF SMALL AMOUNTS OF PCDD AND PCDF IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD N Y ,1982 PP 33-43		X	X								X							

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-MINS	FUR-ANS	DIN MCs	CHEM CHAR	B/T CHEM	TEMP CHEM	HOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAN TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
CAVALLARO,A .ET AL SAMPLING, OCCURRENCE, AND EVALUATION OF PCDDS AND PCDFS FROM INCINERATED SOLID URBAN WASTE CHEMOSPHERE,8:611-631.1980		X	X				X				X							
CHEN,P H .ET AL COMPARATIVE RATES OF ELIMINATION OF SOME INDIVIDUAL POLYCHLORINATED BIPHENYLS FROM THE BLOOD OF PCB-POISONED PATIENTS IN TAIWAN TO BE PUBLISHED IN FOOD CHEM TOXICOL	X												X		X			
CHEN,P H .ET AL LEVELS AND GAS CHROMATOGRAPHIC PATTERNS OF POLYCHLORINATED BIPHENYLS IN THE BLOOD OF PATIENTS AFTER PCB POISONING IN TAIWAN BULL ENVIRON CONTAM TOXICOL.,28:323-328,1982.	X												X		X			
CHEN P H .ET AL POLYCHLORINATED BIPHENYLS, DIBENZOFURANS AND QUATERPHENYLS IN THE TOXIC RICE-BRAN OIL AND PCBS IN THE BLOOD OF PATIENTS WITH PCBS POISONING IN TAIWAN, TO BE PUBLISHED	X		X										X		X			
CHEN,P H ,K I CHANG,AND Y.S LU. POLYCHLORINATED BIPHENYLS AND POLYCHLORINATED DIBENZOFURANS IN THE TOXIC RICE-BRAN OIL THAT CAUSED PCB POISONING IN TAICHUNG BULL ENVIRON CONTAM TOXICOL .26:489-490,1981.	X		X										X		X			
CHESLER,S N .ET AL STANDARD REFERENCE MATERIALS FOR ACCURATE ANALYSES OF PCBS IN OIL ASTM STANDARDIZATION NEWS,10:61:10-20,1982	X										X							

REFERENCE	PCDs	BIO-MING	FUR-ANS	BTN MCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	MEID CHEM	HUM FOX	ANIM TOX	ACCD INCD	ENV IMPT	TONS INFO	TOX/ DISP
CHITTIM,B G.,ET AL CHLORINATED DIBENZOFURANS AND DIBENZO-P-DIOXINS: DETECTION AND QUANTIFICATION IN ELECTRICAL EQUIPMENT AND TOXIC FORMATION DURING THE INCINERATION OF PCBS WELLINGTON SCIENCE ASSOCIATES,DOCKWOOD,ONTARIO,SEPTEMBER 1978.	X	X	X				X				X							X
CHOUDHARY,G OCCUPATIONAL EXPOSURE TO CHLORINATED DIOXINS AND DIBENZOFURANS,A PERSPECTIVE, SUBMITTED FOR PUBLICATION IN ANN ARBOR SCIENCE,ANN ARBOR,MICHIGAN		X	X										X		X			
CHOUDHARY,G G .,AND B HUTZINGER MECHANISTIC ASPECTS OF THE THERMAL FORMATION OF HALOGENATED ORGANIC COMPOUNDS INCLUDING POLYCHLORINATED DIBENZO-P-DIOXINS PART II: THERMOCHEMICAL GENERATION AND DESTRUCTION OF DIBENZOFURANS AND DIBENZO-P-DIOXINE, TOXICOL ENVIRON CHEM .,5:67-82,1982.		X	X		X		X				X							
CHOUDHARY,G G.,ET AL SYNTHESIS OF CHLORINATED DIBENZOFURANS BY PHOTOLYSIS OF CHLORINATED DIPHENYL ETHERS IN ACETONE SOLUTION CHEMOSPHERE,6: 327-332,1977			X			X		X										
CHOUDHARY,G G .,K OLIE,AND B HUTZINGER MECHANISMS IN THE THERMAL FORMATION OF CHLORINATED COMPOUNDS INCLUDING POLYCHLORINATED DIBENZO-P-DIOXINS, IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL .,EDS PERGAMON PRESS,ELMSFORD N Y.,1982 PP,275-301		X					X											

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-RIMS	FUR-ANS	BIN HCs	CHEM CHAR	B/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	DETR CHEM	MAM TOX	AMIB TOX	ACCD INCD	ENV INPI	TRNS INFO	TRT/ DISP
CLEANING OPERATIONS AT THE BINGHAMTON STATE OFFICE BUILDING SUSPENDED NEW YORK STATE PRESS RELEASE, FEB.24, 1981	-	X	X												X			
COCHRANE,W P ,ET AL. ANALYSIS OF TECHNICAL AND FORMULATED PRODUCTS OF 2,4-DICHLOROPHENOXY ACETIC ACID FOR THE PRESENCE OF CHLORINATED DIBENZO-P-DIOXINS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS: IMPACT ON THE ENVIRONMENT. O.HUTZINGER,ET AL.,EDS. PERGAMON PRESS,OXFORD,N Y .1982. PP 209-213.		X		X							X							
COOK,J W SOME CHEMICAL ASPECTS OF POLYCHLORINATED BIPHENYLS (PCBS). ENVIRON HEALTH PERSPECT.,EXPERIMENTAL ISSUE 1:3-12,1972.	X				X						X							
COPELAND,B E ,AND C.E GAMMAGE IMPROVED METHOD FOR POLYCHLORINATED BIPHENYL DETERMINATION IN COMPLEX MATRICES ENVIRON.SCI.TECHNOL.,14:181-184. 1980.	X										X							
COYLE,E DESTRUCTION OF POLYCHLORINATED BIPHENYLS. IN: PROCEEDINGS, 11TH ANNUAL ENVIRONMENTAL SYSTEMS SYMPOSIUM, ARLINGTON, VIRGINIA, NOVEMBER 19-20,1980. AMERICAN DEFENSE PREPAREDNESS ASSOCIATION,ARLINGTON,VA. PP.13-18.	X																	X
CROSBY,D G. CONQUERING THE MONSTER - THE PHOTOCHEMICAL DESTRUCTION OF CHLORODIOXINS IN: DISPOSAL AND DECONTAMINATION OF PESTICIDES N.V. KENNEDY,ED ACS SYMP SER.,73:1-18. 1978.		X						X										X

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MONS 215333

REFERENCE	PCBs	DIB-XINS	FUR-ANS	BTN HCs	CHEM CHAR	B/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	RFG CHEM	ANAL CHEM	METB CHEM	NMR TBX	AMIN TBX	ACCD INCD	ENV INPT	TRNS INFO	TOT/ DISP
CROSBY D G ,AND A S WONG PHOTOCHEMICAL GENERATION OF CHLORINATED DIOXINS CHEMOSPHERE,8:387-230,1979		X						X										
CROSBY,D G ,AND K W WOLLAMEN PHOTODECOMPOSITION OF CHLORINATED BIPHENYLS AND DIBENZO-FURANS BULL ENVIRON CONTAM TOXICOL ,10:370-377,1973	X		X					X										
CROSBY D G ,K W WOLLAMEN AND S S WONG ENVIRONMENTAL GENERATION AND DECRADATION OF DIBENZODIOXINS AND DIBENZOFURANS ENVIRON.HEALTH PERSPEC , EXPERIMENTAL ISSUE 5:209-204,1973		X	X			X		X		X								
CROV K B CUTANEOUS EFFECTS OF CHLORAMNEIGENS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD,N Y ,1982 PP 529-532.	X	X	X	X										X				
CRUMMETT W B ANALYTICAL CHEMISTRY: REVIEW LECTURE TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,SALZBURG,AUSTRIA,OCTOBER 1982		X									X							
CRUMMETT W B ENVIRONMENTAL CHLORINATED DIOXINS FROM COMBUSTION - THE TRACE CHEMISTRIES OF FIRE HYPOTHESIS. IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD N Y ,1982 PP 253-263		X					X				X							
CRUMMETT W B ,AND R N STEHL DETERMINATION OF CHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS IN VARIOUS MATERIALS ENVIRON HEALTH PERSPEC ,EXPERIMENTAL ISSUE 5 15-25,1973		X	X								X							

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIB-XINS	FUR-ANS	OTH HCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	TOXD INFO	TRT/ DISP
CUTIE S S ACCURACY EFFICIENCY OF 2, 3 7 8-TETR.CHLORDBENZA-P-DIOXIN FROM ACT.VE CARBON AND STMED PARTICULATES ANAL CHEM ACTA 123:24-31,1981		X									X							
DECAD,E M ,L B BIRNBAUM,AND M B MATTHEWS DISPOSITION OF 2,3 7,8-TETRACHLORDDIBENZOFURAN IN GUINEA PIGS, RATS AND MONKEYS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS,IMPACT ON THE ENVIRONMENT, O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD,N.Y 1982 PP 307-318			X											X				
DFS ROSSIGO,P E REMEDIAL MEASURES AND DISPOSAL PRACTICES FOR WASTES CONTAINING DIOXINS AND FURANS TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,SALZBURG,AUSTRIA, OCTOBER 1982.		X	X															X
DI DOMENICO A ,E VIVIANO,AND G ZAPPONI MEIHODOLOGICAL PROBLEMS IN ASSESSING 2,3,7,8-TCDD ENVIRONMENTAL CONTAMINATION AT SEVESO IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS,IMPACT ON THE ENVIRONMENT, O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD, N Y ,1982 PP 47-54		X														X		
DI DOMENICO A ,E VIVIANO,AND G ZAPPONI ENVIRONMENTAL PERSISTENCE OF 2,3, 7,8-TCDD AT SEVESO IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL EDS PERGAMON PRESS,ELMSFORD,N Y 1982 PP 105-115		X													X	X		

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-MINS	FUR-AMR	OTH MCs	CHEM CHAR	D/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	MEID CHEM	MAN TOX	AMIN TOX	ACCD INCD	ENV INPT	TRNS INFO	TRT/ DISP
BINGHAM,E J PATE OF BINGHAMTON'S DEALER TOXIC STILL UNCERTAIN NEW YORK TIMES, AUG 10,1981	X														X			
BRYSON,P E ,ET AL DIOXINS, VOL.2: ASSESSMENT OF DIOXIN-FORMING CHEMICAL PROCESSES, EPA-600/2-80-102,INDUSTRIAL ENVIRONMENTAL RESEARCH LABORATORY,CINCINNATI,JUNE 1980.		X			X	X												
BUCKLEY,E J PLANT EMISSIONS: DIOXINS IN PERSPECTIVE: BURNING, INCINERATING, RESOLVING THE ISSUES, SOLID WASTES MANAGE.,64(8):54-57,60-69, 1981.		X			X													
DURFEE,R L ,ET AL PCBs IN THE UNITED STATES: INDUSTRIAL USE AND ENVIRONMENTAL DISTRIBUTION EPA-560/6-76-005, VEASAR, SPRINGFIELD, Va., FEBRUARY 1976.	X															X		
DUVALL,D J ,N A BURGY,AND J A.MESCHER, LABORATORY CHARACTERIZATION OF THE THERMAL DECOMPOSITION OF HAZARDOUS WASTES IN: LAND DISPOSAL OF HAZARDOUS WASTES; PROCEEDINGS OF THE 4TH ANNUAL RESEARCH SYMPOSIUM D B SHARTZ,ED EPA-600/9-78-016,MUNICIPAL ENVIRONMENTAL RESEARCH LABORATORY, CINCINNATI,1978. PP.104-111.				X														X
DUVALL,D S ,AND N A.BURGY LABORATORY EVALUATION OF HIGH-TEMPERATURE DESTRUCTION OF POLYCHLORINATED BIPHENYLS AND RELATED COMPOUNDS EPA-600/2-77-230,MUNICIPAL ENVIRONMENTAL RESEARCH LABORATORY,CINCINNATI,DECEMBER 1977	X	X	X				X											X

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MONS 215336

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-KING	PHR-ANE	BTH HCs	CHEM CHAR	O/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	Env INPT	TANS INFO	TPT/ DISP
CAGGN,D A PARTICULATE CONTAMINATION IN THE BINGHAMTON STATE OFFICE BUILDING PAPER PRESENTED AT PCB SEMINAR, DALLAS,TEXAS,DECEMBER 1981.	X	X	X									—		X	X			
EDISON ELECTRIC INSTITUTE COMMENTS AND STUDIES ON THE USE OF POLYCHLORINATED DIPHENYLS IN RESPONSE TO AN ORDER OF THE UNITED STATES COURT OF APPEALS FOR THE DISTRICT OF COLUMBIA CIRCUIT VOL. 6: POTENTIAL HEALTH EFFECTS IN THE HUMAN FROM EXPOSURE TO POLYCHLORINATED BIPHENYLS AND RELATED IMPURITIES. NEW YORK,FEBRUARY 1982	X		X										X					
ENVIRONMENTAL HEALTH RESOURCES CENTER, UNIVERSITY OF ILLINOIS, CHICAGO POLYCHLORINATED BIPHENYLS: HEALTH EFFECTS AND RECOMMENDATIONS IIEC 76/08,ILLINOIS INSTITUTE FOR ENVIRONMENTAL QUALITY,CHICAGO,OCTOBER 1976.	X											X	X	X	X	X		
EPA PAVES WAY FOR PCB BURNING. EPA J.,OCT:ES-22,1982.	X																	X
EOM,S D.,B.L.TAYLOR,AND T.D.TIEDMAN DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN RESIDUES ON METAL SURFACES BY GC-MS CHEMOSPHERE,8: 7-14,1978.		X									X							
SOWE,K THE PHENOXY ACID PROBLEM IN SWEDEN AND A POSSIBLE DIOXIN PROBLEM - A REVIEW IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS P.CATTABENI,A.CAVALLARO,AND G.GALLI,EDS SPECTRUM PUBLICATIONS,NEW YORK,1978. PP 101-103		X		X														

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOXINS	FURANS	OTHER HCs	CHEM CHAR	O/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCR	ENV INPT	TOXS INFO	IDI/ DISP
ESPOSITO,M P ,ET AL DIOXINS. VOL 1: SOURCES, EXPOSURE, TRANSPORT AND CONTROL EPA-600/2-80-198,INDUSTRIAL ENVIRONMENTAL RESEARCH LABORATORY,CINCINNATI,JUNE 1980		x			x	x			x	x			x					x
ESPOSITO M P ,T.O TIERNAN,AND F E DRYDEN DIOXINS EPA-600/2-80-197,INDUSTRIAL ENVIRONMENTAL RESEARCH LABORATORY,CINCINNATI,NOVEMBER 1980		x			x	p			x		x		x		x			x
FARA,S.M ,ET AL CHLORACNE OFTER RELEASE OF TCDD AT SEVESO, ITALY IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS, ELMSFORD,N.Y .1982. PP 349-369		x											x		x			
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FIRESTONE,D THE 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN PROBLEM:A REVIEW IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL,ED ECOL. BULL (STOCKHOLM),27:39-52,1978.		x									x		x			x		
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REFERENCE	PCBs	BIO-MINS	FUR-ANS	BTH NCs	CHEM CHAR	B/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	IRMS INFO	INT/ DISP
FREUDENTHAL,J. THE QUANTITATIVE DETERMINATION OF TCDD WITH DIFFERENT MASS SPECTROMETRIC METHODS IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS, F CATTABENI,A CAVALLARO, AND G GALLI,EDS SPECIRUM PUBLICATIONS,NEW YORK,1978. PP.43-49		x									x								
GARA,A STUDIES OF POLYCHLORINATED DIBENZOFURANS PH D.DISSERTATION, UNIVERSITY OF UMEA,SWEDEN,1980			x		x						x		x		x				
GARA A ,ET AL SYNTHESIS OF HALOGENATED BIPHENYL ETHERS AND DIBENZOFURANS - A DISCUSSION OF SPECIFIC ISOMERS AVAILABLE CHEMOSPHERE,10/3&8-388,1981			x	x	x														
GARA,A.,ET AL SYNTHESIS OF HIGHER CHLORINATED DIBENZOFURANS CHEMOSPHERE,8:480-484,1979			x		x														
GARATTINI,S ,ET AL IMMUNOSUPPRESSANT ACTIVITY OF TCDD IN MICE IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS. PERGAMON PRESS,ELMSFORD,N Y.,1982 PP 483-489		x												x					
GARVEY,C. CONTROLLING PCB'S - A NEW APPROACH EPA J ,7(7):29,1981	x																	x	
GE AGREES TO STUDY PCB CONTAMINATION OF HOUSATONIC BY 170 FITCHFIELD,MASS PLANT HAZARD WASTE NEWS ,3(30):330, JULY 13,1981	x															x			

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOXINS	FURANS	BIN HCs	CHEM CHAR	P/C CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	META CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/DISP
GIAM,C S ,H S CHAN,AND G S NEFF RAPID AND INEXPENSIVE METHOD FOR DETECTION OF POLYCHLORINATED BIPHENYLS AND PHTHALATES IN AIR ANAL.CHEM ,47,2319-2320,1975	X										X							
GIERTHY,J F ,AND D D CRANE A PRELIMINARY REPORT ON THE EVALUATION OF AN IN VITRO ASSAY FOR THE DETECTION OF "DIOXIN-LIKE" ACTIVITY USING EXTRACTS OF SOOT FROM THE BINGHAMTON STATE OFFICE BUILDING NEW YORK STATE DEPT.OF HEALTH,ALBANY,1982		X										X		X				
GOLDMANN,P J SCHWERSTE AKUTE CHLORAKNE DURCH TRICHLORPHENOL-ZERSETZUNGSPRODUKTE ARBEITSMED SOZIALMED.PRAEVENTIVMED ,7:12-18,1972.		X		X									X		X			
GOLDSTEIN,J.A.ET AL 2,3,7,8-TETRACHLORODIBENZOFURAN IN A COMMERCIALLY AVAILABLE 99% PURE POLYCHLORINATED BIPHENYL ISOMER IDENTIFIED AS THE INDUCER OF HEPATIC CYTOCHROME P-448 AND ARYL HYDROCARBON HYDROXYLASE IN THE RAT. DRUG METAB.DISPOS.,6:258-264,1978.	X		X									X						
GREENLEE,W F.,AND A POLAND NUCLEAR UPTAKE OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN C57BL/6J AND DBA/2J MICE: ROLE OF THE HEPATIC CYTOSOL RECEPTOR PROTEIN J BIOL CHEM ,254:9814-9821,1979.		X										X		X				
GREENLEE,W F ,AND A POLAND TCDD: A MOLECULAR PROBE FOR INVESTIGATING THE INDUCTION OF ARYL HYDROCARBON HYDROXYLASE IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND G GALLI, SPECTRUM PUBLICATIONS,NEW YORK,1978. PP 113-121		X											X					

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REFERENCE	PCBs	DIOXINS	FURANS	BINCs	CHEM CHAR	O/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TOT/DISP
GREIG, J B, AND E OSBORNE CHANGES IN RAT HEPATIC CELL MEMBRANES DURING 2,3,7,8-TETRACHLORO-DIBENZO-P-DIOXIN INTOXICATION. IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI, A CAVALLARO, AND G GALLI, EDS SPECTRUM PUBLICATIONS, NEW YORK, 1978 PP.105-111.		X												X				
GRAY, A P, ET AL SYNTHESIS OF SPECIFIC POLYCHLORINATED DIBENZO-P-DIOXINS. J.ORG CHEM, 41:2428-2437, 1976.		X			X													
GROSS, M L, ET AL METHOD VALIDATION FOR THE DETERMINATION OF TETRACHLORODIBENZODIOXIN AT THE LOW PARTS PER TRILLION LEVEL ANAL CHEM., 53:1902-1906, 1981		X									X							
HAILE, C.L, AND E.BALADI. METHODS FOR DETERMINING THE POLYCHLORINATED BIPHENYL EMISSIONS FROM INCINERATION AND CAPACITOR AND TRANSFORMER FILLING PLANTS EPA-600/4-77-048, MIDWEST RESEARCH INSTITUTE, KANSAS CITY, MO, NOVEMBER 1977	X										X						X	
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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-XING	FUR-ANG	BIM MCs	CHEM CHAR	G/I CHEM	VEMP CHEM	PHOT CHEM	ENV CHEM	AFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPI	TRNS INFO	TOI/ DISP
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HARLESS R L .AND D G DOUGLAS LOW- AND HIGH-RESOLUTION GAS-CHROMATOGRAPHY-MASS SPECTROMETRY (GC-MS) METHOD OF ANALYSIS FOR THE PRESENCE OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) IN ENVIRONMENTAL SAMPLES IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F COTTRENI,A CAVALLARO,AND C GALLI, EDS SPECTRUM PUBLICATIONS,NEW YORK, 1978 PP 31-47		X									X							
HARLESS,R L .AND D G LEWIS. QUANTITATIVE DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN ADSORBED BY GAS CHROMATOGRAPHY/MASS SPECTROMETRY IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL.,EDS PERGAMON PRESS,ELMSFORD,N.Y .,1982 PP 25-36		X									X							
HARLESS,R L.,ET AL SAMPLE PREPARATION AND GAS CHROMATOGRAPHY-MASS SPECTROMETRY DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN ANAL CHEM .53:1329-1340,1980		X									X							
HARRIS,M W .ET AL. GENERAL BIOLOGICAL EFFECTS OF TCDD IN LABORATORY ANIMALS ENVIRON HEALTH PERSPEC .SPECIAL ISSUE 5:101-109,1973		X												X				

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOXINS	FURANS	BTH MCs	CHEM CHAR	B/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MUR TOX	AMIN TOX	ACCD INCD	ENV IMPI	ENS INFO	TBT/ DISP
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HAUSEN,B DIPHENYL METABOLISM BY RAT LIVER MICROSOMES. REGIOSELECTIVE EFFECTS OF INDUCERS, INHIBITORS, AND SOLVENTS DRUG METAB DISPOS.,9:810-817,1981	X											X		X				
HAY A THE CHEMICAL DETECTION OF DIOXIN PLENUM PRESS, NEW YORK,1982 PP 187-205.		X													X			
HAY,A U H EXPOSURE TO TCDD: THE HEALTH RISK TO CHLORINATED DIBENZENE AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT D HUTZINGER ET AL.,EDS PERGAMON PRESS,ELMSFORD,N.Y ,1982 PP 289-299.		X											X		X			
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HELLING,C S ,AND D C TURNER PESTICIDE MOBILITY:DETERMINATION BY SOIL THIN-LAYER CHROMATOGRAPHY. SCIENCE, 162:562-563,1968				X					X		X							
HELSING L S DESTROYING DIOXIN: A UNIQUE APPROACH WASTE AGE,11(10):40-43,1980		X						X										X

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TOXS INFO	TRT/DISP
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HIGUCHI, K. PCB POISONING AND POLLUTION. KODANSHA, TOKYO; ACADEMIC PRESS, NEW YORK, 1976.	x		x										x					
HOLDEN, A.V., AND K. MARSDEN. SINGLE STAGE CLEANUP OF ANIMAL TISSUE EXTRACTS FOR ORGANOCHLORINE RESIDUE ANALYSIS. J. CHROMATOGR., 44: 481-492, 1969	x	x	x	x							x							
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HOMBERGER, E., ET AL. THE SEVESO ACCIDENT: ITS NATURE, EXTENT, AND CONSEQUENCES. ANN. OCCUP. HYG., 22: 327-370, 1979		x													x			
HUETTER, R., AND M. PHILIPPI. STUDIES ON MICROBIAL METABOLISM OF TCDD UNDER LABORATORY CONDITIONS. IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT. O. HUTZINGER, ET AL., EDS. PERGAMON PRESS, ELMSFORD, N.Y., 1982. PP 87-92		x										x						

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOX-INS	FUR-ANS	OTH-ERS	CHEM CHAR	P/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFC CHEM	ANAL CHEM	METE CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	EMRG INFO	TRT/DISP
HUFF, J E ET AL LONG-TERM HAZARDS OF POLYCHLORINATED DIBENZODIOXINS AND POLYCHLORINATED DIBENZOFURANS ENVIRON HEALTH PERSPEC., 36:221-240, 1980.		x	x		x								x	x	x			
HUGHES, B M ,ET AL ANALYTICAL METHODS FOR HERBICIDE ORANGE VOL I. DETERMINATION OF CHEMICAL COMPOSITION. REPORT NO 10 78-0115,U S AIR FORCE SYSTEMS COMMAND,ANDREWS AFB,WASHINGTON,D C ,MAY 1978.		x									x							
HUTZINGER,O ,ET AL PHOTOCHEMICAL DEGRADATION OF DI- AND OCTACHLORODIBENZOFURAN ENVIRON HEALTH PERSPEC.,EXPERIMENTAL ISSUE 5:267-271,1973			x					x										
HUTZINGER,O ,ET AL. POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS: A BIOANALYTICAL APPROACH CHEMOSPHERE,10:19-25,1981.		x	x				x				x							
HUTZINGER,O ,S SAFE,AND V ZITKO ANALYSIS OF CHLORINATED AROMATIC HYDROCARBONS BY EXHAUSTIVE CHLORINATION QUALITATIVE AND STRUCTURAL ASPECTS OF THE PERCHLORO-DERIVATIVES OF BIPHENYL, NAPHTHALENE, TERPHENYL, DIBENZOFURAN, DIBENZODIOXIN, AND DDE. INT J. ENVIRON ANAL.CHEM., 2:95-106,1972.		x	x	x							x							
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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOXINS	FURANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/DISP
ISENSEE, A R BIOACCUMULATION OF 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN. IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL, ED ECOL BULL. (STOCKHOLM), 27:255-262, 1978		X							X			X						
JANSSON B AND G SUNDSTROM FORMATION OF POLYCHLORINATED DIBENZOFURANS (PCDF) DURING A FIRE ACCIDENT IN CAPACITORS CONTAINING POLYCHLORINATED BIPHENYLS (PCB) IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER, ET AL., EDS PERGAMON PRESS, ELMSFORD, N.Y., 1982 PP 201-207	X		X			X	X								X		X	
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KEARNEY P C, ET AL ENVIRONMENTAL SIGNIFICANCE OF CHLORODIOXINS IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR, ED ADV CHEM SER, 120:105-111, 1973		X				X		X	X							X		

[illegible] OF REFERENCES FOR PCBs

REFERENCE	DIB-KINS	FUR-ANS	BIM HCB	CHEM CHAR	B/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMIN TOX	ACCD INCD	ENV IMPT	IRNS INFO	TRT/ DISP
KEARNEY P C ,ET AL (TETRACHLORODIBEN-ZODIOXIN IN THE ENVIRONMENT:SOURCES, FATE, AND DECONTAMINATION ENVIRON HEALTH PERSPEC ,EXPERIMENTAL ISSUE 5: 253-277 1973	X							X	X								
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KHERA,K S ,AND J A GRADICK POLYCHLORODIBENZO-P-DIOXINS: PERINATAL EFFECTS AND THE DOMINANT LETHAL TEST IN WISTAR RATS IN: CHLORODIOXINS - ORIGIN AND FATE E H BLOBIN,ED ADV CHEM SER ,120:78-84,1973	X												X				
KIMBLE,H J COMMENTS ON 'CHLORINATED DIBENZO-P-DIOXINS AND RELATED COMPOUNDS IN INCINERATOR EFFLUENTS' CHEMOSPHERE,10:243-244,1981	X									X							
KIMBROUGH,R D ,ET AL EPIDEMIOLOGY AND PATHOLOGY OF A TETRACHLORODIBENZODIOXIN POISONING EPISODE, ARCH ENVIRON.HEALTH,32:77-86,1977	X												X	X			
KING,H.E ,A H.SNEPHER,AND H A GUTEE CARCINOGENESIS BIOASSAY OF CHLORINATED DIBENZODIOXINS AND RELATED CHEMICALS ENVIRON HEALTH PERSPEC ,EXPERIMENTAL ISSUE 5:163-170,1973	X												X				
KLOPFER,A D ,ET AL OCCURRENCE OF TCDD IN ENVIRONMENTAL SAMPLES FROM SOUTHWEST MISSOURI PRESENTED AT THE ACS SYMPOSIUM,KANSAS CITY, MISSOURI,SEPTEMBER 1983	X							X		X							

REFERENCE	PCBs	BIO-XING	FUR-ANS	MTH MCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMIN TOR	ACCB CHCB	ENV IMPT	TOHS INFO	TOI/ GIVP
KHUTSON,J C .AND A POLAND 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN: TOXICITY IN VIVO AND IN VITRO IN: TOXICOLOGY OF HALOGENATED HYDROCARBONS: HEALTH AND ECOLOGICAL EFFECTS, A S.O EHAN AND H H STANTON,EDS. PERGAMON PRESS, NEW YORK,1981. PP.187-881.		X												X				
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KOPP,F E. PCB DISPOSAL, DECLORINING, AND TREATMENT. IN: CONFERENCE PROCEEDINGS: NATIONAL CONFERENCE ON POLYCHLORINATED BIPHENYLS (NOVEMBER 1975.CHICAGO,ILLINOIS),EPA-560/6-75-004.MARCH 1976 PP 180-183	X																	X

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-MIM	FUR-ANS	OTH PCBs	CHEM CHAR	D/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD TOXD	ENV IMPT	TRNS INFO	IDT/DISP
##:EDEL H THE BIGHING: TOXIC AND STILL TROUBLESOME ENVIRONMENT,23: 6-13,1981		X							X	X			X		X			
KUEHL,D W UNUSUAL POLYHALOGENATED CHEMICAL RESIDUES IDENTIFIED IN FISH TISSUE FROM THE ENVIRONMENT CHEMOSPHERE,FR:231-243,1981		X	X	X										X		X		
KUWATSUKA,H ,ET AL. YUSHO, A POISONING CAUSED BY RICE OIL CONTAMINATED WITH POLYCHLORINATED BIPHENYLS. NAGASAKI MEDICAL REP .,46:1063-1091,1971	X		X										X		X			
KUWATSUKA,H ,Y NAGUMO,AND J NAGATOMO SOME OF THE RECENT FINDINGS CONCERNING THE YUSHO. IN: CONFERENCE PROCEEDINGS: NATIONAL CONFERENCE ON POLYCHLORINATED BIPHENYLS (NOVEMBER 1975, CHICAGO,ILLINOIS) EPA-560/6-75-004, MARCH 1976, PP 16-29	X	X											X					
KUROKI,H.,AND Y NAGUMO DETERMINATION OF POLYCHLORINATED DIBENZOFURANS (DBFS) OBTAINED IN PATIENTS WITH YUSHO CHEMOSPHERE,7:771-777,1978	X		X			X							X					
KUWABARA,K.,ET AL. LEVELS OF POLYCHLORINATED BIPHENYLS IN BLOOD OF UNBORN-FED CHILDREN WHOSE MOTHERS ARE NON-OCCUPATIONALLY EXPOSED TO PCBS. BULL.ENVIRON CONTAM TOXICOL.,21: 490-462,1979	X												X					
LAFORNARA,J P CLEANUP AFTER SPILLS OF TOXIC SUBSTANCES J WATER POLLUT CONTROL FED .,50:617-627,1978	X														X			X

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-SYNS	FUR-ANS	OTH MCs	CHEM CHAR	S/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METE CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	TRNS INFO	DISP
LAMPARSKI L L ,AND T J NESTRICK DETERMINATION OF TETRA- HEXA-, HEPTA- AND OCTACHLORODIBENZO-P-DIOXIN ISOMERS IN PARTICULATE SAMPLES AT PART PER TRILLION LEVELS ANAL CHEM ,52: 2045-2054,1980		X									X							
LAMPARSKI,L L ,AND T J.NESTRICK OPTIMIZATION PROCEDURE FOR SYNTHESIS OF HIGHER CHLORINATED DIBENZO-P-DIOXINS FROM 1,2,3,7,8-PENTACHLORODIBENZO-P-DIOXIN ANAL. CHEM .,54:482-486,1982		X			X													
LAMPARSKI L L.,AND T J NESTRICK SYNTHESIS AND IDENTIFICATION OF THE 10 HEXACHLORODIBENZO-P-DIOXIN ISOMERS BY HIGH PERFORMANCE LIQUID AND PACKED COLUMN GAS CHROMATOGRAPHY CHEMOSPHERE,10:3-10,1981.		X									X							
LAMPARSKI,L L ,AND T J NESTRICK THE ISOMER-SPECIFIC DETERMINATION OF TETRACHLORODIBENZO-P-DIOXIN AT PART PER TRILLION CONCENTRATIONS IN: CHLORINATED DIPHENYL AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL .EDS PERGAMON PRESS,ELMSFORD,N Y .1982 PP 1-14		X									X							
LAMPARSKI,L L.,T.J NESTRICH,AND D K DIEHL DETERMINATION OF PART-PER-TRILLION CONCENTRATIONS OF 2 3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN FISH ANAL CHEM .51:1493-1498,1969		X									X							
LANGER H G ,ET AL THERMAL CHEMISTRY OF CHLORINATED PHENOLS IN:CHLORODIOXINS - ORIGIN AND FATE E H BLAIR, ED ADV CHEM SER 120:26-38,1973		X		X			X											

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOXINS	FURANS	OTH HCs	CHEM CHAR	P/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	REG/DISP
LANGER, H G, T P BRADY AND P R BRIGGS FORMATION OF DIBENZODIOXINS AND OTHER CONDENSATION PRODUCTS FROM CHLORINATED PHENOLS AND DERIVATIVES ENVIRON HEALTH PERSPEC, EXPERIMENTAL ISSUE 5:3-7, 1973.		X				X												
LANGHORST, M L, AND L A SHADOFF DETERMINATION OF PARTS-PER-TRILLION CONCENTRATIONS OF TETRA-, HEXA-, HEPTA-, AND OCTACHLORODIBENZO-P-DIOXINS IN HUMAN MILK ANAL CHEM, 52: 2037-2044, 1980		X									X		X					
LIBERTI, A, AND D BROCCO FORMATION OF POLYCHLORODIBENZODIOXINS AND POLYCHLORODIBENZOFURANS IN URBAN INCINERATOR EMISSIONS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL, EDS PERGAMON PRESS, ELMSFORD, N Y 1982 PP 245-251		X	X				X											
LIBERTI, A ET AL FIELD PHOTODEGRADATION OF TCDD BY ULTRA-VIOLET RADIATIONS IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI, A CAVALLARO AND G GALLI, EDS SPECTRUM PUBLICATIONS, NEW YORK, 1978 PP 195-199		X						X										
LIHACH N MANAGING PCB S EPRI J., 6 (8):23-26, 1981	X										X		X					X
LINDAHL, R, C RAPPE, AND H R BUSER FORMATION OF POLYCHLORINATED DIBENZOFURANS (PCDFS) AND POLYCHLORINATED DIBENZO-P-DIOXINS (PCDDS) FROM THE PYROLYSIS OF POLYCHLORINATED DIPHENYL ETHERS CHEMOSPHERE 9:351-361, 1980		X	X				X											

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIB-XINS	FUR-ANS	OTH HCs	CHEM CHAR	O/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	NFC CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMTH TOX	ACCD INCD	ENV IMPT	TONS INFO	TOT/ DISP
LINNAINMAA K. NON-MUTAGENICITY OF PHENOXY-ACID HERBICIDES 2,4-D AND MCPA PRESENTED AT THE ACS DIOXIN SYMPOSIUM, KANSAS CITY, MISSOURI, SEPTEMBER 1982				X								X		X				
LLOYD J W ,ET AL POLYCHLORINATED BIPHENYLS. J OCCUP MED ,18:109-113,1976	X				X					X			X	X				
LUSTENHOUWER,J.W A .K OLIE,AND O.HUTZINGER CHLORINATED DIBENZO-P-DIOXINS AND RELATED COMPOUNDS IN INCINERATOR EFFLUENTS CHEMOSPHERE,9:(5):501-522, 1980.		X	X				X				X							
MAHLE,N H ,AND L F WHITTING THE FORMATION OF CHLORODIBENZO-P-DIOXINS BY OIL ONIDATION AND CHLORINATION OF BITUMINOUS COAL CHEMOSPHERE,9:893-898, 1980		X					X											
MASUDA,Y ,AND H KUROKI. POLYCHLORINATED DIBENZOFURANS AND RELATED COMPOUNDS IN PATIENTS WITH "YUSHO". IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS. PERGAMON PRESS, ELMSFORD,N Y ,1982. PP 561-568	X		X										X					
MASUDA,Y ,H KUROKI,AND J NAGAYAMA. POLYCHLORINATED DIBENZOFURANS IN THE TISSUES OF PATIENTS WITH YUSHO AND THEIR ENZYME INDUCING ACTIVITIES ON ARYL HYDROCARBON HYDROXYLASE PRESENTED AT THE ACS DIOXIN SYMPOSIUM, KANSAS CITY, MISSOURI, SEPTEMBER 1982.			X									X	X					

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIB-KINS	FUR-ANS	OTH RCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMIR TOX	ACCD INCD	ENV INPT	TRNS INFO	TRT/ DISP
MATSUMURA,F .AND H J BENEZET STUDIES ON THE BIOACCUMULATION AND MICROBIAL DEGRADATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN ENVIRON HEALTH PERSPEC..EXPERIMENTAL ISSUE 5: 253-258,1973		X										X				X		
MATTHEWS,H B .AND H ANDERSON PCB CHLORINATION VERSUS PCB BIOTRANSFORMATION AND EXCRETION. IN: CONFERENCE PROCEEDINGS: NATIONAL CONFERENCE ON POLYCHLORINATED BIPHENYLS (NOVEMBER 1975,CHICAGO,ILLINOIS) EPA-560/6-75-004,MARCH 1976 PP 88-94	X													X				
MATTHIASSON B. SURVEY ABOUT TOXICOLOGICAL DATA OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND G.GALLI), 605 SPECTRUM PUBLICATIONS,NEW YORK, 1978 PP 133-138		X												X				
MAZES,T.,ET AL. CHARACTERIZATION OF TETRACHLORODIBENZODIOXINS *PREPRINT EXTENDED ABSTRACT * PRESENTED BEFORE THE DIVISION OF ENVIRONMENTAL CHEMISTRY AMERICAN CHEMICAL SOCIETY,KANSAS CITY,MISSOURI,SEPTEMBER 1982.			X							X								
MCCONNELL,E E .AND J.A BRAND THE TOXICOPATHOLOGY OF TCDD IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND G GALLI, SPECTRUM PUBLICATIONS,NEW YORK,1978 PP 137-141.		X												X				

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-KINS	FUR-ANS	OTH HCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/DISP
MCGRAW,M D THE PCB PROBLEM:SEPARAT-ING FACT FROM FICTION. ELECTR.WORLD, 197(2):49-72,1983.	X	X	X								X		X		X	X	X	X
MCKINNEY,J ,AND E MCCONNELL STRUC-TURAL SPECIFICITY AND THE DIOXIN RECEPTOR IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE EN-VIRONMENT O HUTZINGER,ET AL ,EDS. PERGAMON PRESS,ELMSFORD,N.Y.,1980 PP 367-381.		X										X		X				
MCKINNEY,J ,ET AL DEVELOPMENT AND RELIABILITY OF A RADIOIMMUNOASSAY FOR 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN. IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS. PERGAMON PRESS,ELMSFORD,N Y ,1982 PP 67-75		X									X							
MCKINNEY,J D ANALYSIS OF 2,3,7,8-TE-TRACHLORODIBENZO-PARA-DIOXIN IN EN-VIRONMENTAL SAMPLES. IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C. RAMEL,ED ECOL.BULL (STOCKHOLM),27: 53-66,1978.		X							X		X							
MCNULTY,W P ,I H POMERANTZ,AND T.J FARRELL CHRONIC TOXICITY OF 2,3,7,8-TETRACHLORODIBENZOFURAN FOR RHESUS MACAQUES IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT. O.HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD, N Y ,1982 PP 411-417.			X											X				
MCNULTY,W P ,ET AL PERSISTENCE OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN FAT OF A RHESUS MONKEY (MACACA MULATTA) FOOD CHEM (IN PRESS)		X										X		X				

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	BIPH NCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	NFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCS INCD	ENV IMPT	TRNS INFO	REG/DISP
MERCIER,M.,ED. CRITERIA (DOSE/EFFECT RELATIONSHIPS) FOR ORGANOCHLORINE PESTICIDES:REPORT OF A WORKING GROUP OF EXPERTS PUBLISHED FOR THE COMMISSION OF THE EUROPEAN COMMUNITIES BY PERGAMON PRESS,OXFORD,1981. PP.329-381.		X			X				X	X		X	X	X	X			
MILLER G A .L.A.NORRIS,AND C.L.HAWKES TOXICITY OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) IN AQUATIC ORGANISMS ENVIRON.HEALTH PERSPECT., EXPERIMENTAL ISSUE 5:177-186,1973.		X												X		X		
MILLER,S DESTROYING HAZARDOUS WASTES ENVIRON.SCI TECHNOL .15:1268-1269,1981.	X	X																X
MILLER,S THE PCB IMBROGLIO ENVIRON SCI TECH.,17:11A-14A,1983.																		
MILLER,S THE PERSISTENT PCB PROBLEM ENVIRON SCI.TECHNOL ,16:98A-99A, 1982	X																	X
MILNES M H FORMATION OF 2,3,7,8-TETRACHLORODIBENZODIOXIN BY THERMAL DECOMPOSITION OF SODIUM 2,4,5-TRICHLOROPHENATE. NATURE,232:395-396,1971.		X				X	X								X			
MITCHUM,R K .ET AL INITIAL REPORT, VALIDATION STUDY FOR THE GC-ATMOSPHERIC IONIZATION-MS METHOD FOR THE ISOMER-SPECIFIC DETERMINATION OF 2,3,7,8-TCDD PRESENTED AT THE NCB DIOXIN SYMPOSIUM, KANSAS CITY, MISSOURI,SEPTEMBER 1983.		X									X							

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIB-KINS	FUR-ANS	BIN MCs	CHEM CHAR	B/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMIN TOX	ACCB INCD	ENV IMPI	IRNS INFO	TRT/ DISP
MITCHUM R K ,W A KORFMACHER,AND G F MOLER ATMOSPHERIC PRESSURE IONIZATION MASS SPECTROMETRY. THE ISOMER SPECIFIC DETERMINATION OF TRACE LEVELS OF 2,3,7,8-TCDD IN THE PRESENCE OF CONTAMINANTS PRESENTED AT 30TH ANNUAL MEETING,AMER;CAN SOCIETY FOR MASS SPECTROMETRY,MINNEAPOLIS, 1981		X									X							
NIYATA N ,AND I KAGAMOTO INVESTIGATION ON ORGANO-CHLORINATED COMPOUNDS FORMED IN KANEMI RICE OIL THAT CAUSED THE "YUSHO." J.FOOD HYG.SOC JAPAN, 20:1-9,1979	X		X										X		X			
NIYATA,N ,I KAGAMOTO,AND N KUNITA DETECTION AND DETERMINATION OF UNKNOWN ORGANOCHLORINATED COMPOUNDS IN KANEMI RICE OIL CAUSED THE "YUSHO " J FOOD HYG SOC JAPAN,19:364-371,1978	X		X								X		X		X			
MOLES,G F ,ET AL, CONFIDENCE LIMITS FOR THE ISOTOPE DILUTION/GAS CHROMATOGRAPHIC-MASS SPECTROMETRIC DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN ENVIRONMENTAL SAMPLES PRESENTED AT THE ACS DIOXIN SYMPOSIUM KANSAS CITY,MISSOURI,SEPTEMBER 1982		X									X							
MOORE,J A IONICITY OF 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN. IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL,ED ECOL BULL (STOCKHOLM),27:134-144 1978		X												X				
MOORE,J A ,D K GUPTA,AND J G VOS TOXICITY OF 2,3,7,8-TETRACHLORODIBENZO-FURAN - PRELIMINARY RESULTS IN: CONFERENCE PROCEEDINGS: NATIONAL CONFERENCE ON POLYCHLORINATED BIPHENYLS (NOVEMBER 1975 CHICAGO, ILLINOIS) EPA-560/6-75-004,MARCH 1976 PP 77-98			X											X				

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MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	DIB-KINS	FUR-ANS	BTH HCp	CHEM CHAR	O/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	HFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	IRNS INFO	IRT/ DISP
ADOPE J A ,ET AL COMPOSATIVE TOXICITY OF THPEE HALOGENATED DIBENZOFURANS IN GUINEA PIGS, MICE, AND RHESUS MONKEYS ANN N Y ACAD SCI.,320:151-163,1979			X											X				
ADOPE,J A ,ET AL PRENATAL EFFECTS OF MATERNAL EXPOSURE TO 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) ENVIRON HEALTH PERSPEC ,EXPERIMENTAL ISSUE 5:81-85,1973		X												X				
ADRITA,M ,ET AL. DETAILED EXAMINATION OF POLYCHLORINATED DIBENZOFURANS IN PCB PREPARATIONS AND RANDOM YUSHO OIL BULL ENVIRON CONTAM TOXICOL.,19:67-73,1977	X		X										X		X			
ADRITA,M ,J NAKAGAWA,AND C DOPPE. POLYCHLORINATED DIBENZOFURAN (PCDF) FORMATION FROM PCB MIXTURE BY HEAT AND OXYGEN BULL ENVIRON CONTAM TOXICOL ,19:668-676,1978	X		X				X											
ADRITA,M J NAKAGAWA,AND R.ARIYAMA FORMATION OF POLYCHLORINATED DIBENZOFURANS IN ULLMAN REACTION, BULL.ENVIRON CONTAM TOXICOL ,19:200-204 1977			X															
ADRAN,M ,B.SUNDSTROEM,AND C A WACHTMEISTER. 2,3,7,8-TETRACHLORODIBENZOFURAN, A CRITICAL BYPRODUCT IN THE SYNTHESIS OF 2,2',4,4',5,5'-HEXACHLOROBIPHENYL BY THE ULLMAN REACTION ACTA CHEM SCAND ,27:3181-3183,1973	X		X		X					X								
ADSELBY,C L ,C L GERACI,AND J BURG POLYCHLORINATED BIPHENYL EXPOSURE IN TRANSFORMER MAINTENANCE OPERATIONS AM IND HYG ASSOC J ,43:170-174,1982	X	X	X														X	

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	A/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	RFC CHEM	ANAL CHEM	META CHEM	HUM TOX	ANIM TOX	ACCO INCO	ENV IMPT	TRNS INFO	DIS/DISP
MUELDER,W W ,AND L A SHADOFF THE PREPARATION OF UNIFORMLY LABELED (14) C-2,7-DICHLORODIBENZO-P-DIOXIN AND (14)C-2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR,ED ADV CHEM,SER 120:1-6,1973.		X								X								
NAKAYAMA,J ,M KURATSUNE,AND Y MASUDA DETERMINATION OF CHLORINATED DIBENZOFURANS IN KANECHLORS AND "YUSHO OIL" BULL ENVIRON CONTAM TOXICOL , 15:9-13,1976.	X		X								X							
NARISADA,M INFRARED ABSORPTION SPECTRA OF AROMATIC ETHER COMPOUNDS. II CHARACTERISTIC BANDS OF DIBENZO-P-DIOXIN DERIVATIVES YAKUGAKU ZASSHI,79: 183-186,1959		X									X							
NATIONAL ELECTRICAL MANUFACTURERS ASSOCIATION REPLY COMMENTS ON NOTICE OF PROPOSED RULEMAKING CONCERNING POLYCHLORINATED BIPHENYLS (PCBS): USE IN ELECTRICAL EQUIPMENT. WASHINGTON, D C.,JUNE 1982.	X		X														X	
NATIONAL INSTITUTES OF HEALTH. CARCINOGENESIS BIOASSAY OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN SWISS-WEBSTER MICE NTP-80-32,BETHESDA,MD , FEBRUARY 1982		X												X				
NATIONAL RESEARCH COUNCIL OF CANADA POLYCHLORINATED DIBENZO-P-DIOXINS: CRITERIA FOR THEIR EFFECTS ON MAN AND HIS ENVIRONMENT OTTAWA,1981		X			X	X		X	X			X	X	X				

REFERENCE	PCDDs	DIOXINS	FURANS	OTH HCs	CHEM CHAR	P/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	META CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/DISP
NATIONAL RESEARCH COUNCIL OF CANADA POLYCHLORINATED DIBENZO-P-DIOXINS: LIMITATIONS TO CURRENT ANALYTICAL TECHNIQUES OTTAWA,1981.		x									x							
NAU,H ,R BASS,AND D NEUBERT. TRANSFER OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) TO THE MOUSE EMBRYO,FETUS, AND NEONATE IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS. IMPACT ON THE ENVIRONMENT O.HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD, N Y., 1982. PP.319-330		x												x				
NEAL,R A ,AND P BEATTY INFLUENCE OF ENZYME INDUCTION ON TCDD TOXICITY. IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO, AND G.GALLI,EDS. SPECTRUM PUBLICATIONS,NEW YORK,1978 PP 143-156		x												x				
NEAL,R A ,P W BEATTY,AND T.A GASIEWICZ STUDIES OF THE TOXICITY OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) ANN N.Y ACAD SCI ,320:204-213,1979			x									x		x				
NEBERT,D W ,AND H V GELBOIN SUBSTRATE-INDUCIBLE MICROSOMAL ARYL HYDROXYLASE IN MAMMALIAN CELL CULTURE. J BIOL.CHEM,243:6242-6249,1968				x								x						
NESTRICK,T ,L LAMPARSKI,AND R.TOWNSEND, CHLOROPHENATE PYROLYTIC SYNTHESIS OF THE 22 TETRACHLORODIBENZO-P-DIOXIN ISOMERS WITH STRUCTURAL CHARACTERIZATION VIA PHOTOLYTIC PROPERTIES IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS,IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS. PERGAMON PRESS,ELMSFORD,N Y ,1982 PP 37-46		x						x			x							

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-RING	FOR-AND	OTH HCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	MEID CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TOT/ DISP
MEDICK,T J ,L L LAMPARSKI,AND A N DIEHL SYNTHESIS AND IDENTIFICATION OF THE 22 TETRACHLORODIBENZO-P-DIOXIN ISOMERS BY HIGH PERFORMANCE LIQUID CHROMATOGRAPHY AND GAS CHROMATOGRAPHY ANAL.CHEM .51:1639-1651.1979.		X									X							
NEUBERT,D .ET AL A SURVEY OF THE BIOLOGICAL EFFECTS OF TCDD IN MAMMALIAN SPECIES ENVIRON HEALTH PERSPEC . EXPERIMENTAL TOXIC,5:67-78,1973		X												X				
NILSSON,C A STUDIES OF POLYCHLORINATED 2-PHENOXYPHENOLS,PRECURSORS OF CHLORINATED DIBENZO-P-DIOXINS. PH.D THESIS,UNIVERSITY OF UMEA. 30 ED UMEA,SWEDEN,1977.		X			X													
NILSSON,C.A .AND L RENBERG FURTHER STUDIES ON IMPURITIES IN CHLORO-PHENOLS J.CHROMATOGR .99:325-333, 1974		X		X	X						X							
NILSSON,C.A.,ET AL CHROMATOGRAPHIC EVIDENCE FOR THE FORMATION OF CHLORODIBENZOFURANS FROM CHLORO-2-PHENOXYPHENOLS J CHROMATOGR .90:137-147,1974		X				O	R	X			X							
NISBET,I C T CRITERIA DOCUMENT FOR PCBS EPA-440/9-76-021, MASSACHUSETTS AUDUBON SOCIETY,LINCOLN,MASS 1976	X		X		X			X	X			X	X	X		X		
NORSTROM, A , K ANDERSSON, AND C SAPPE FORMATION OF CHLORODIBENZOFURANS BY IRRADIATION OF CHLORINATED DIPHENYL ETHERS CHEMOSPHERE,8:81-84, 1976			X		X													

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIOXINS	FURANS	DIR CHEM	CHAR CHEM	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	REIB CHEM	NUM IDX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
NORSTROM,A ,ET AL SYNTHESIS OF CHLORINATED DIBENZOFURANS AND CHLORINATED ANISOL-DIBENZOFURANS FROM THE CORRESPONDING DIPHENYL ETHERS AND NITRO-DIPHENYL-ETHERS CHEMOSPHERE,8:331-343, 1979			X		X							-						
NORSTROM,R ,K ANDERSSON,AND C RAPPE, STUDIES ON THE FORMATION OF CHLORODIBENZOFURANS BY IRRADIATION OR PYROLYSIS OF CHLORINATED DIPHENYL ETHERS CHEMOSPHERE,6:241-248,1977			X		X		X											
O KEEFE,P W A NEUTRAL CLEANUP PROCEDURE FOR ICDD RESIDUES IN ENVIRONMENTAL SAMPLES. IN: SIGNIN: ION:COLOGICAL AND CHEMICAL ASPECTS F.CATTABENI,A CAVALLARO,AND G GALLI,EDS SPECTRUM PUBLICATIONS,NEW YORK,1978 PP 59-77		X									X							
O KEEFE,P W IRACE CONTAMINANTS IN A POLYCHLORINATED DIPHENYL FIRE DETARDANT AND A SEARCH FOR THESE COMPOUNDS IN ENVIRONMENTAL SAMPLES BULL ENVIRON CONTAM TOXICOL 22:420-428, 1979	X	X	X	X					X									
OLIE K ,J W A LINTELMANNEA,AND O HUTZINGER POLYCHLORINATED DIBENZO-P-DIOXINS AND RELATED COMPOUNDS IN INCINERATOR EFFLUENTS IN:CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL ,EDS PERGAMON PRESS,ELMSFORD, N Y ,1982 PP 227-241		X					X				X							

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	SYN MCH	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	DIST/DISP
OLIE K ,P L VERMEULEN,AND O HUTZINGER CHLORODIBENZO-P-DIOXINS AND CHLORODIBENZOFURANS ARE TRACE COMPONENTS OF FLY ASH AND FLUE GAS OF SOME MUNICIPAL INCINERATORS IN THE NETHERLANDS CHEMOSPHERE,6:455-459,1977		x	x				x											
SHIOMI,M A ,AND W C TRENCH PCBS IN PERSPECTIVE INDUS WASTES,27(2):33-30,1981.	x		x												x			
OUW,H K ,G R SIMPSON,AND D S SIYALI. USE AND HEALTH EFFECTS OF AROCLOR 1242, A POLYCHLORINATED BIPHENYL, IN AN ELECTRICAL INDUSTRY ARCH ENVIRON HEALTH,3:189-194,1976	x												x		x			
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PAASIVIRTA,J ,ET AL. ON THE LIMIT OF DETECTION OF TCDD IN GAS CHROMATOGRAPHY CHEMOSPHERE,6:355,1977		x									x							
PEAKALL,D B POLYCHLORINATED BIPHENYLS: OCCURRENCE AND BIOLOGICAL EFFECTS RESIDUE REV ,44:1-21,1972	x								x			x	x	x				
PETTY,J D.,ET AL THE COMPOSITION OF PCDF AND PCDD RESIDUES IN SEDIMENTS OF THE HUDSON AND HOUSATONIC RIVERS PRESENTED AT THE ACS DIOXIN SYMPOSIUM KANSAS CITY,MISSOURI,SEPTEMBER 1982		x	x						x									

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-KINS	FWD-ANS	OTH MCs	CHEM CHAR	O/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMIN TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
PHILIPPI,M .ET AL FATE OF TCDD IN MICROBIAL CULTURE AND IN SOIL UNDER LABORATORY CONDITIONS PRESENTED AT 12TH FEBS SYMPOSIUM,ZURICH,SWITZERLAND,SEPTEMBER 1980		X										X						
PIPER,W W ,J G WAGE,AND P.J GEHRING. INCRETION AND TISSUE DISTRIBUTION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN THE RAT IN: CHLORODIOXINS - ORIGIN AND FATE E.H BLAIR,ED ADV.CHEM SER ,120:68-81,1973.		X												X				
PLIMMER,J R, PHOTOLYSIS OF TCDD AND TRIFLURALIN ON SILICA AND SOIL. BULL ENVIRON.CONTAM TOXICOL ,20:87-92, 1978		X					X		X									
PLIMMER,J R ,ET AL PHOTOCHEMISTRY OF DIBENZO-P-DIOXINS IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR, ED ADV CHEM.SER ,120:44-64,1973		X					X											
POCCHIARI,F 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN DECONTAMINATION IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS, C RAMEL,ED ECOL.BULL. (STOCKHOLM),27:67-78,1978		X			X										X			X
POCCHIARI,F CONTINGENCY PLANNING FOR AND RESPONSE TO EMERGENCIES AND ACCIDENTS INVOLVING TOXIC MATERIALS. TO BE PRESENTED AT THE 2ND INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,SALZBURG,AUSTRIA,OCTOBER 1982				X											X			

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	DIS-KINS	FUR-ANS	BIN MCs	CHEM CHAR	D/T CHEM	TERP CHEM	PHOT CHEM	ENV CHEM	RFG CHEM	ANAL CHEM	METD CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
POCCHIARI F ,V SILANO,AND A ZAPPIERI HUMAN HEALTH EFFECTS FROM ACCIDENTAL RELEASE OF TETRACHLORODIBENZO-P-DIOXIN (TCDD) AT SEVESO, ITALY ANN N Y ACAD SCI .320:311-325,1979		X											X					
POHL R J ,AND J A FOUTS A RAPID METH FOR ASSAYING THE METABOLISM OF 7-ETHOXYRESORUFIN BY MICROSOMAL SUBCELLULAR FRACTIONS ANAL BIOCHEM .107, 150-155,1980				X								X						
POHLAND A E ,AND D C TANG PREPARATION AND CHARACTERIZATION OF CHLORINATED DIBENZO-P-DIOXINS J AGRIC. FOOD CHEM .26,1093-1099,1978		X			X						X							
POIGER H ,AND C SCHLATTER BIOLOGICAL DEGRADATION OF TCDD IN SOIL NATURE. 281,706-707,1979		X										X		X				
POIGER,H ,AND C SCHLATTER INFLUENCE OF SOLVENTS AND ADSORBENTS ON DERMAL AND INTESTINAL ABSORPTION OF TCDD FOOD COSMET TOXICOL .18:477-481,1980		X										X						
POIGER H ET AL STRUCTURE ELUCIDATION OF MAMMALIAN TCDD-METABOLITES PRESENTED AT 2D INTERNATIONAL WORKSHOP ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,ARLINGTON,VIRGINIA, OCTOBER 1981		X										X		X				
POIGER,H ET AL STRUCTURE ELUCIDATION OF MAMMALIAN TCDD-METABOLITES EXPERIENTIA.36:484-486,1980		X										X		X				

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-KINS	FUR-ans	BIN CHEM HCs	CHAR	B/S CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
POIGER,H ,H WEBER AND C SCHLATTER SPECIAL ASPECTS OF METABOLISM AND KINETICS OF TCDD IN DOGS AND RATS ASSESSMENT OF TOXICITY OF TCDD-METABOLITE(S) IN GUINEA PIGS IN:CHLORINATED DIOXINS AND RELATED COMPOUNDS,IMPACT ON THE ENVIRONMENT O HUTZINGER, ET AL ,EDS PERGAMON PRESS,ELMSFORD, N Y ,1982. PP 317-333			X									X		X				
POLAND A ,AND E GLOVER 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN AND ENZYME INDUCTION IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL,ED ECOL BULL (STOCKHOLM),27: 149-148,1978			X									X		X				
POLAND,A ,AND E GLOVER CHLORINATED DIPHENYL INDUCTION OF ARYL HYDROCARBON HYDROXYLASE ACTIVITY:A STUDY OF THE STRUCTURE-ACTIVITY RELATIONSHIP MOL PHARMACOL.,12:328-338,1977	X											X						
POLAND A AND E GLOVER CHLORINATED DIBENZO-P-DIOXINS:POTENT INDUCERS OF D-AMINOLEVULIC ACID SYNTHETASE AND ARYL HYDROCARBON HYDROXYLASE. II. A STUDY OF THE STRUCTURE-ACTIVITY RELATIONSHIP MOL PHARMACOL.,9:736-747, 1973			X									X						
POLAND A ,AND E GLOVER COMPARISON OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN A POTENT INDUCER OF ARYL HYDROCARBON HYDROXYLASE WITH 3-METHYLCHOLANTHRENE MOL PHARMACOL.,10:349-359, 1974			X									X						

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-KINS	FUE-ANS	OTH HCs	CHEM CHAR	S/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METD CHEM	NUM TOX	AMIN BOX	ACCD INCD	ENV TRPT	TRNS INFO	TAT/ DISP
POLAND A ,AND E GLOVER STUDIES ON THE MECHANISM OF TOXICITY OF THE CHLORINATED DIBENZO-P-DIOXINS ENVIRON HEALTH PERSPEC ,EXPERIMENTAL ISSUE 5:245-251.1973		X										X		X				
POSITIVE TECHNOLOGIES,INC CLASS NOTES FOR ONE-DAY COURSE ON PCB REGULATIONS CITY OF INDUSTRY,CALIFORNIA,JANUARY 1982	X																X	X
POWERS W J ASSESSMENT AND CONTROL OF POLYCHLORINATED BIPHENYLS (PCBS). IN: PROCEEDINGS, 11TH ANNUAL ENVIRONMENTAL SYSTEMS SYMPOSIUM, ARLINGTON, VA , NOVEMBER 19-20,1980 AMERICAN DEFENSE PREPAREDNESS ASSOCIATION, ARLINGTON,VA PP 8-11	X																	X
PRESTON D D ,AND J D ALLEN 2,8 ,5,5 -TETRACHLOROBIPHENYL, ISOLATION AND IDENTIFICATION OF METABOLITES GENERATED BY RAT LIVER MICROSOMES DRUG METAB DISP ,8:197-203,1980.	X											X						
RAMEL,C CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS: CHEMISTRY--SUMMARY IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C.RAMEL,ED ECOL BULL (STOCKHOLM),27:29-17,1978		X	X		X	X		X										
RAMEL,C CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS: CONCLUSIONS AND RECOMMENDATIONS IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL,ED ECOL BULL (STOCKHOLM),27: 13-16,1978		X			0				X									

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	META CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TANK INFO	TOT/DISP
RAPPE,C E 3 7,8-TETRACHLORODIBEN-ZO-P-DIOXIN (TCDD) - INTRODUCTION IN DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND G GALLI,EDS SPECTRUM PUBLICATIONS. NEW YORK,1978 PP 8-11.		X													X			
RAPPE,C CHEMICAL BACKGROUND OF THE PHENOXY ACIDS AND DIOXINS IN: CHLO-RINATED PHENOXY ACIDS AND THEIR DIO-XINS C RAMEL,ED ECOL BULL.(STOCK-HOLM) 27:28-30,1978		X			X													
RAPPE,C. DECONTAMINATION OF PRODUCTS FORMED DURING THE INDUSTRIAL PRE-PARATION OF 2,4,5-TRICHLOROPHENOL IN: DIOXIN: TOXICOLOGICAL AND CHEMI-CAL ASPECTS F.CATTABENI,A.CAVALLARO, AND G GALLI,EDS. SPECTRUM PUBLICA-TIONS,NEW YORK,1978. PP.179-182.		X				X				X								
RAPPE,C ERFARENHETER FRAN PCB-BRAN-DER. SKANDIA-TIPS,17(4):17-18,1982	X		X								X				X			
RAPPE,C MAELSORIGHER VID ARBETE MED NY OCH ANVAEND PCB UMEA UNIVERSI-TAET,UMEA,SWEDEN,1982	X		X										X				X	
RAPPE,C. REPORT ON THE ANALYSIS OF BINGHAMPTON (SIC) SOOT. MAY 1981.		X	X												X			
RAPPE,C UNIVERSITY OF UMEA PROCE-DURES FOR ANALYSIS OF PCDDS AND PCDFS IN FLY ASH PERSONAL COMMUNICATION, NOVEMBER 1982		X	X								X							
RAPPE,C ,A GARA,AND H R BUSER. IDEN-TIFICATION OF POLYCHLORINATED DIBEN-ZOFURANS (PCDFS) IN COMMERCIAL CHLO-ROPHENOL FORMULATIONS CHEMOSPHERE, 7:981-991,1978			X	X							X							

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	DIB-XINS	FUR-ANS	OTH HCs	CHEM CHAR	O/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	TRNS INFO	TRI/ BIOP
&APPE C .AND C A NILSSON AN ARTI-FACT IN THE GAS CHROMATOGRAPHIC DE-TERMINATION OF IMPURITIES IN PENTA-CHLOROPHENOL J CHROMATOGR.,67:247-253,1972		X		X	X						X							
&APPE C .AND H H &UNGE CHEMICAL PRO-PERTIES AND ANALYTICAL METHODS. IN: HALOGENATED BIPHENYLS,TERPHENYLS, NAPHTHALENES, DIBENZODIOXINS AND RE-LATED PRODUCTS D D.E]NORDBERG,ED ELSEVIER/NORTH HOLLAND BIOMEDICAL PRESS,AMSTERDAM,1980 PP.41-76	X	X	X	X	X		X				X							
&APPE C .AND H D &UNGE POLYCHLORINA-TED DIOXINS AND DIBENZOFURANS IN INCINERATOR EFFLUENTS. I:1981		X	X				X											
&APPE,C .AND R HUTZEN CHEMICAL ANAL-YSIS OF HUMAN SAMPLES: IDENTIFICATION AND QUANTIFICATION OF POLYCHLORINATED DIBENZO AND DIBENZOFURANS PRESENTED AT THE U S -SWEDISH WORKSHOP,MAY 1982		X	X								X		X					
&APPE,C .ET AL ANALYSIS OF POLYCHLO-RINATED DIBENZOFURANS IN YUSHO OIL USING HIGH RESOLUTION GAS CHROMATO-GRAPHY-MASS SPECTROMETRY CHEMO-SPHERE,6:23)-236,1977.	X		X		X						X							
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&APPE,C .ET AL FORMATION OF POLY-CHLORINATED DIBENZO-P-DIOXINS (PCDDS) AND DIBENZOFURANS (PCDFS) BY BURNING OR HEATING CHLOROPHENOLES CHEMO-SPHERE,7:269-901,1978		X	X				X											

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	MAM TRX	AMIN TOX	ACCD INCD	ENV IMPT	TRNS INFO	IRI/ DISP
RAPPE,C .ET AL IDENTIFICATION OF POLYCHLORINATED DIBENZOFURANS (PCDFS) RETAINED IN PATIENTS WITH YUSHO CHEMOSPHERE,8:259-266,1979			X								X		X					
RAPPE C .ET AL IDENTIFICATION OF POLYCHLORINATED DIBENZOFURANS IN ENVIRONMENTAL SAMPLES NATURE 281: 534-536,1981			X								X					X		
RAPPE C .ET AL IDENTIFICATION OF POLYCHLORINATED BIPHENYLS (PCBS) AND DIBENZOFURANS (PCDFS) IN HUMAN SAMPLES: OCCUPATIONAL EXPOSURE AND YUSHO PATIENTS PRESENTED AT 2D INTERNATIONAL WORKSHOP ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,ARLINGTON,VIRGINIA,OCTOBER 1981		X	X								X		X		X			
RAPPE C .ET AL OCCUPATIONAL EXPOSURE TO POLYCHLORINATED DIOXINS AND DIBENZOFURANS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL .EDS PERGAMON PRESS,ELMSFORD,N Y .1982 PP 499-513		X	X										X					
RAPPE C .ET AL. POLYCHLORINATED DIOXINS, DIBENZOFURANS AND OTHER POLYNUCLEAR AROMATICS FORMED DURING INCINERATION AND PCB FIRES *PREPRINT EXTENDED ABSTRACT * PRESENTED BEFORE THE DIVISION OF ENVIRONMENTAL CHEMISTRY, AMERICAN CHEMICAL SOCIETY,KANSAS CITY,MISSOURI,SEPTEMBER 1982	X	X	X				X											
RAPPE C .ET AL POLYCHLORINATED DIOXINS (PCDDS), DIBENZOFURANS (PCDFS) AND OTHER POLYNUCLEAR AROMATICS (PCP-MAS) FORMED DURING PCB FIRES CHEM SCRIPTS 20:64-67,1982	X	X	X	X			X											X

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	IRT/DISP
RAPPE,C ,H R BUSER AND H P BOSSHARDT, DIOXINS, DIBENZOFURANS, AND OTHER POLYHALOGENATED AROMATICS: PRODUCTION USE FORMATION, AND DESTRUCTION ANN N Y ACAD SCI ,320:1-18,1979	x	x	x	x	x	x				x								
RAPPE,C ,H R BUSER AND H P BOSSHARDT IDENTIFICATION AND QUANTIFICATION OF POLYCHLORINATED DIBENZO-P-DIOXINS (PCDDS) AND DIBENZOFURANS (PCDFS) IN 2 4 5-T-ESTER FORMULATIONS AND HERBI-CIDE ORANGE CHEMOSPHERE,7:431-438, 1978		x	x								x							
RAPPE C ,S MARKLUND,AND P A BERG-QVIST ERFAGENHETER FRAN MAGRA PCB-BRAENDER PERSONAL COMMUNICATION, 1982	x	x					x								x			
REGGIANI,G HUMAN OBSERVATIONS; RE-VIEW LECTURE TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLO-RINATED DIOXINS AND RELATED COM-POUNDS,SALZBURG AUSTRIA,OCTOBER 1982		x											x					
REGGIANI G TOXICOLOGY OF TCDD AND RELATED COMPOUNDS: OBSERVATIONS IN MAN IN: CHLORINATED DIOXINS AND RE-LATED COMPOUNDS, IMPACT ON THE EN-VIRONMENT O HUTZINGER,ET AL EDS PERGAMON PRESS,ELMSFORD,N Y ,1982 PP 463-493	x	x	x	x									x					
RENBERG L ,G SUNDSTROM AND K SUNDH-NYGARD PARTITION COEFFICIENTS OF OR-GANIC CHEMICALS DERIVED FROM REVERSED PHASE THIN LAYER CHROMATOGRAPHY; EVALUATION OF METHODS AND APPLICATION ON PHOSPHATE ESTERS POLYCHLORINATED PARAFFINS AND SOME PCB SUBSTITUTES CHEMOSPHERE 9 683-691,1980	x										x							

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REFERENCE	PCBs	BIO-XINS	FUR-ANS	DIM NCs	CHEM CHAR	Q/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METR CHEM	HUM TOX	ANIM TOX	ACCD IN-S	ENV IMPT	IRNS INFO	TRI/ DISP
ARSING CLARGE ABOUT PCB S ENVIRON SCI TECHNOL ,10:122-122 1976	X												X				X	
ROACH J A G .AND I H PORERANTZ THE FINDING OF CHLORINATED DIBENZOFURANS IN A JAPANESE POLYCHLORINATED BI-PHENYL SAMPLE BULL ENVIRON CONTAM TOXICOL ,12:338-342,1974	X		X								X							
BOBERTSON L W ET AL ARYL HYDROCAR-BON HYDROXYLASE (AHH) INDUCTION BY POLYCHLORINATED BIPHENYLS (PCBS): EN-HANCEMENT BY PHOTOLYSIS TOXICOLOGY, 22:103-114,1981	X											X		X				
RYAN J J ,AND J C PILON CHLORINATED -DIBENZODIOXINS AND -DIBENZOFURANS IN CHICKEN LIVER AND LIVERS ARISING FROM PENTACHLOROPHENOL CONTAMINATION OF WOOD SHAVINGS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT E HUTZINGER,ET AL EDS PERGAMON PRESS,ELMSFORD,N Y 1982 PP 183-186		X	X											X				
RYAN J J ET AL A SURVEY OF LAKE ON-TARIO FISH FOR 2,3,7,8-TETRACHLOROBI-BENZO-P-DIOXIN DERIVATIVE *PREPRINT EXTENDED ABSTRACT * PRESENTED BEFORE THE DIVISION OF ENVIRONMENTAL CHEMIS-TRY AMERICAN CHEMICAL SOCIETY,KANSAS CITY,MISSOURI,SEPTEMBER 1982		X												X				
SAFE,S BIOCHEMICAL EFFECTS, REVIEW LECTURE TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINA-TED DIOXINS AND RELATED COMPOUNDS SALZBURG,AUSTRIA,OCTOBER 1982		X										X						

REFERENCE	PCBs	BIO-XENS	FUR-ANS	BTN MCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	META CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	IRMS INFO	IRT/ BIOP
BAPS O ,ET AL PCBS AS MICROSOMAL ENZYME INDUCERS: STRUCTURE-ACTIVITY STUDIES IN: TOXICOLOGY OF HALOGENATED HYDROCARBONS: HEALTH AND ECOLOGICAL EFFECTS H A A KHAN AND D N STANTON, EDS PERGAMON PRESS NEW YORK,1981 PP 104-119	X											X						
BAFF S ET AL PHOTODECOMPOSITION OF HALOGENATED AROMATIC COMPOUNDS IN: IDENTIFICATION AND ANALYSIS OF POLLUTANTS IN WATER L H KEITH,ED ANN ARBOR SCIENCE,ANN ARBOR,MICH.,1976 PP 30-47.	X							X										
BAFE S H ET AL PCBS AS ANH INDUCERS IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD,N Y ,1982. PP 383-391.	X											X		X				
BAINT-AUF,A THE STRUCTURE AND BIOCHEMICAL EFFECTS OF TCDD IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F COTTRODENI,A CAVALLARO,AND C GALLI EDS SPECTRUM PUBLICATIONS, NEW YORK,1978 PP 187-161.		X										X		X				
BAMBEIN,J THE REVERS INCIDENT TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,SALZBURG,AUSTRIA, OCTOBER 1982		X													X			
BMPERSTEIN H O O J GARBON,AND E J FAERER PCB CONTAMINATION IN DISTRIBUTION TRANSFORMERS J.Environ Sci Health,A17:241-251,1982	X																X	

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	S/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	RFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV INPT	TRNS INFO	TRT/ DISP
RAMYEN,T ,ET AL BIOANALYSIS OF POLYCHLORINATED DIBENZOFURAN AND DIBENZO-P-DIOXIN MIXTURES IN FLY ASH PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,SALZBURG,AUSTRIA, OCTOBER 1982		X	X								X	X						
GENECIN,A CONTAMINATION OF AN OFFICE BUILDING WITH PCDD, FURANS, DIOXINS, AND DIPHENYLENES AFTER AN ELECTRICAL TRANSFORMER EXPLOSION IN BINGHAMTON, NEW YORK TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS,SALZBURG,AUSTRIA,OCTOBER 1982	X	X	X	X											X		X	
SCHLATTER C.,AND H POIGER ANIMAL TOXICITY; REVIEW LECTURE TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS, SALZBURG,AUSTRIA, OCTOBER 1982		X												X				
SCHWETZ,B A ,ET AL TOXICOLOGY OF CHLORINATED DIBENZO-P-DIOXINS IN: CHLORODIOXINS - ORIGIN AND FATE, E H BLAIR,ED ADV CHEM SER ,120:95-109, 1973		X												X				
SCHWETZ,B A ,ET AL TOXICOLOGY OF CHLORINATED DIBENZO-P-DIOXINS. ENVIRON HEALTH PERSPEC ,EXPERIMENTAL ISSUE 5:87-99,1973		X												X				
SHADOFF,L A THE DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN HUMAN MILK IN: PESTICIDE ANALYTICAL METHODOLOGY J HARVEY,JR ,AND G ZWEIG,EDS ACS SYMP SER ,136:877-885, 1980		X									X							

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-XINS	FURANS	OTHER HCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	REFS CHEM	ANAL CHEM	METH CHEM	MASS TOM	ORIN ION	ACCD INCD	ENV INFT	TRNS INFO	TRT/ DISP
SHADOFF,L A ,AND S A.RUNNEL THE DETERMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN IN BIOLOGICAL EXTRACTS BY GAS CHROMATOGRAPHY MASS SPECTROMETRY BIOMASS MASS SPECTROM . 5:7-13,1978		X									X							
SHANNAHAN,C E ,ET AL CHLOROLYSIS APPLIED TO THE CONVERSION CHLOROCARBON RESIDUES EPA-600/2-78-146, HOECHST-UHDE CORPORATION,ENGLEWOOD CLIFFS,N J ,JULY 1978.				X														X
SHAUB W M ,AND W TSANG. PHYSICAL AND CHEMICAL PROPERTIES OF DIOXINS IN RELATION TO THEIR DISPOSAL (DRAFT) CHEMICAL KINETICS DIVISION,CENTER FOR CHEMICAL PHYSICS,NATIONAL BUREAU OF STANDARDS,WASHINGTON,D.C.		X			X	X	X											X
SHIAPLS,N P ,AND S S SAYLES BIOTRANSFORMATION OF PCB IN NATURAL ASSEMBLAGES OF FRESHWATER MICROORGANISMS ENVIRON SCI TECHNOL.,14:367-369,1980	X								X			X						
SMITH,L M ,AND J L JOHNSON EVALUATION OF INTERFERENCES FROM REVIEW SERIES OF POLYCHLORINATED AROMATIC COMPOUNDS IN AN ANALYTICAL METHOD FOR POLYCHLORINATED DIBENZOFURANS AND DIOXINS PRESENTED AT THE ACS DIOXIN SYMPOSIUM,KANSAS CITY,MISSOURI,SEPTEMBER 1980		X	X	X							X							
SMITH L M.,ET AL IDENTIFICATION AND QUANTITATION OF POLYCHLORODIBENZOFURANS IN ENVIRONMENTAL SAMPLES PRESENTED AT 29TH ANNUAL MEETING,AMERICAN SOCIETY FOR MASS SPECTROMETRY, MINNEAPOLIS,1981			X								X					X		

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REFERENCE	PCBs	BIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/I CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TXT/ DISP
SMITH,R N .ET AL ANALYSIS FOR 0,3,7, 8-TETRACHLORODIBENZOFURAN AND 2,3,7, 8-TETRACHLORODIBENZO-P-DIOXIN IN A SOOT SAMPLE FROM A TRANSFORMER EXPLOSION IN BINGHAMTON NEW YORK CHEMOSPHERE,11:719-720,1982		X	X								X				X		X	
BOLCH J G .ET AL GC-MS PROCEDURES FOR DETERMINATION OF CHLORODIBENZO-P-DIOXIN ISOMERS IN PARTICULATES FROM COMBUSTION SOURCES PRESENTED AT 20TH ANNUAL MEETING,AMERICAN SOCIETY FOR MASS SPECTROMETRY,MINNEAPOLIS,1981		X									X							
SPACIE,A .J L HAMELINK,AND E C MAYGRANT APPLICATION OF AN EVAPORATIVE LOSS MODEL TO ESTIMATE THE PERSISTENCE OF CONTAINANTS IN LENTIC ENVIRONMENTS IN: AQUATIC TOXICOLOGY AND HAZARD EVALUATION F.L.MAYER AND J L HAMELINK,EDS. ASTM STP 634,AMERICAN SOCIETY FOR TESTING AND MATERIALS PHILADELPHIA,1977 PP 214-227.				X					X									
STALLING,D L..ET AL ISOLATION AND ANALYSIS OF POLYCHLORINATED DIBENZO-FURANS IN AQUATIC SAMPLES. IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT. O HUTZINGER,ET AL .EDS PERGAMON PRESS,ELMSFORD,N Y .1982 PP 77-90		X	X								X					X		
STALLING,D L .ET AL RESIDUES OF POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS IN LAWRENTIAN GREAT LAKES FISH, PRESENTED AT 2D INTERNATIONAL WORKSHOP ON CHLORINATED DIOXINS AND RELATED COMPOUNDS, ARLINGTON,VIRGINIA.OCTOBER 1981		X	X						X					X		X		

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-KINS	FUR-ANS	OTH HCs	CHEM CHAR	D/T CHEM	IEMP CHEM	PHOT CHEM	ENV CHEM	AFC CHEM	ANAL CHEM	METB CHEM	MAM TOX	AMIR TOX	ACCD INCD	ENV IMPT	TRNS INFO	IRT/ RISP
STANLEY J S ,ET AL SAMPLING AND ANALYSIS PROTOCOL FOR ASSESSING ORGANIC EMISSIONS FROM STATIONARY COMBUSTION SOURCES IN EXPOSURE EVALUATION DIVISION COMBUSTION STUDIES EPA-560/3-82-014 MIDWEST RESEARCH INSTITUTE, KANSAS CITY MO JANUARY 1982	x	x	x								x				x			
STEHL R H AND L L LAMPARSKI CONDUSTION OF SEVERAL 2,4,5-TRICHLOROPHENOXY COMPOUNDS: FORMATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN SCIENCE 197:1998-1999,1977		x		x			x												
STEHL R H ,ET AL THE STABILITY OF PENTACHLOROPHENOL AND CHLORINATED DIOXINS TO SUNLIGHT, HEAT, AND COMBUSTION IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR,ED ADV CHEM SER ,120:119-125,1973		x				x	x	x	x										
STAIK,J J U A TCDD AND COGNATE HEPATIC PORPHYRIA IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD,N Y 1982 PP 313-317		x											x						
SUNDSTROM G 3,3 4,4 -TETRACHLOROAZOBENZENE AND 3,3',4,4'-TETRACHLOROAZOXYBENZENE POTENT CHLORACNEGENS AND ENZYME INDUCERS - AN OVERVIEW IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS IMPACT ON THE ENVIRONMENT O HUTZINGER ET AL ,EDS PERGAMON PRESS,ELMSFORD,N Y 1982 PP 337-353				x		x						x	x						

REFERENCE	DIO-XINS	FUR-ANS	OTH PCDs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METH CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/ DISP
TAYLOR J S ,AND K H LLOYD CHLORINATED FROM 2,2 4,4 -TETRACHLOROAZOXYBENZENE AND 3,3',4,4'-TETRACHLOROAZOBENZENE; UPDATE AND REVIEW IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT O HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD N Y ,1982 PP 539-543			x									x					
TAYLOR,M L ,ET AL. ADDENDUMS OF INCINERATION PROCESSES AS SOURCES OF SUPERTOXIC CHLORINATED HYDROCARBONS: CONCENTRATIONS OF CDDS/CDFS AND POSSIBLE PRECURSOR COMPOUNDS IN INCINERATOR EFFLUENTS PRESENTED AT THE ACS DIOXIN SYMPOSIUM,KANSAS CITY,MISSOURI,SEPTEMBER 1982	x	x				x											
TAYLOR,M L ,ET AL LEVELS OF TETRACHLORO-DIBENZO-P-DIOXIN IN ENVIRONMENTAL AND BIOLOGICAL SAMPLES AS DETERMINED BY GAS CHROMATOGRAPHY-HIGH RESOLUTION MASS SPECTROMETRY PRESENTED AT 24TH ANNUAL MEETING,AMERICAN SOCIETY FOR MASS SPECTROMETRY,SAN DIEGO,1976	x									x					x		
THOMPSON,L J OFF-SITE TRANSPORT OF 2,3,7,8 TETRACHLORODIBENZO-P-DIOXIN FROM A PRODUCTION-DISPOSAL FACILITY PRESENTED AT THE ACS DIOXIN SYMPOSIUM KANSAS CITY,MISSOURI,SEPTEMBER 1983	x								x								x
TIERNAN,T O CHLORODIBENZODIOXINS AND CHLORODIBENZOFURANS; AN OVERVIEW IN: DETECTION OF HAZARDOUS WASTES J EKNED,ED ANN ARBOR SCIENCE ANN ARBOR MICHIGAN,1982 PP 243-261	x	x			x		x			x	x	x					

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MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FURANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFC CHEM	ANAL CHEM	METH CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	INNS INFO	TXT/ DISP
TIEDMAN,T D ,ET AL. ANALYTICAL METHODOLOGY FOR DETERMINATION OF CHLORODIBENZOTOXINS AND CHLORODIBENZOFURANS IN FLY ASH AND STACK EFFLUENTS FROM MUNICIPAL WASTE INCINERATORS PRESENTED BEFORE THE DIVISION OF ENVIRONMENTAL CHEMISTRY,AMERICAN CHEMICAL SOCIETY,LAS VEGAS,MARCH 1982			X	X			X				X							
TIEDMAN,T D.,ET AL. CHARACTERIZATION OF TOXIC COMPONENTS IN THE EFFLUENTS FROM A REFUSE-FIRED INCINERATOR, RESOUR CONSERV.,8:343-354,1982.			X	X			X											
TIEDMAN,T D ,ET AL. DIOXINS. VOL.2: ANALYTICAL METHOD FOR INDUSTRIAL WASTES. EPA-600/2-80-197,INDUSTRIAL ENVIRONMENTAL RESEARCH LABORATORY, CINCINNATI,JUNE 1980.			X								X							
TIEDMAN,T D ,ET AL. INCINERATION OF CHEMICAL WASTES CONTAINING POLYCHLORINATED BIPHENYLS: DEMONSTRATION OF TESTS CONDUCTED AT ROLLINS ENVIRONMENTAL DEVICES, DEER PARK, TEXAS AND ENERGY SYSTEMS COMPANY, EL DORADO, ARKANSAS IN: DETOXIFICATION OF HAZARDOUS WASTES / ERNEST,ED. ANN ARBOR SC IENCE,ANN ARBOR,MICHIGAN, 1982 PP 143-184	X		X	X			X				X							
TOSINE,H DIOXINS: A CANADIAN PERSPECTIVE PRESENTED AT THE ACS DIOXIN SYMPOSIUM KANSAS CITY,MISSOURI,SEPTEMBER 1982.			X															
TOWNSEND D I THE USE OF DIOXIN TOXNER GROUP ANALYSIS TO IDENTIFY SOURCES AND DEFINE BACKGROUND LEVELS OF DIOXINS IN THE ENVIRONMENT;A REVIEW,UPDATE, AND EXTENSION OF THE PRESENT TWOMEY IN: CHLORINATED DIOXINS AND RELATED COMPOUNDS, IMPACT ON THE ENVIRONMENT D HUTZINGER,ET AL ,EDS PERGAMON PRESS,ELMSFORD, N.Y ,1982 PP.269-274.			X			X	X												

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-XINS	FUR-ANS	OTH CHEM MCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	HEIB CHEM	HUM TOX	AMIN TOX	ACCD INCD	ENV IMPT	INNS INFO	TRT/ DISP
TSANG,W ,AND W BOWARD. CHEMICAL PROCESSES IN THE INCINERATION OF HAZARDOUS MATERIALS CHEMICAL KINETICS DIVISION,CENTER FOR CHEMICAL PHYSICS, NATIONAL BUREAU OF STANDARDS,WASHINGTON,D C		X					X											X
TOROKHATO,O ,F MATSUMURA,AND O HASE FATE OF 2,3,7,8-TCDD IN AN OUTDOOR POND AND IN MODEL AQUATIC ECOSYSTEMS ENVIRON TOXICOL CHEM ,1:61-68,1982.		X				N			O					X		X		
TULP H T.,AND O HUTZINGER, RAT METABOLISM OF POLYCHLORINATED BIPHENYLS-P-BIPHENYL CHEMOSPHERE,7:751-760,1978.		X										X		X				
TULP,O T H ,O SCHMITZ,AND O.HUTZINGER THE BACTERIAL METABOLISM OF 4,4' DICHLOROBIPHENYL AND ITS SUPPRESSION BY ALTERNATIVE CARBON SOURCES. CHEMOSPHERE,8:193-198,1978.	X											X						
TURNER,J H ,AND O.O COLLINS, BIRMINGHAM STATE OFFICE BUILDING SHORT-INDUCED MORPHOLOGIC ALTERATIONS IN THE LIVERS OF GUINEA PIGS NEW YORK STATE DEPT OF HEALTH,ALBANY,1982	X	X												X	X			
U O ENVIRONMENTAL PROTECTION AGENCY CRITERIA AND STANDARDS DIVISION. 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN: AMBIENT WATER QUALITY CRITERIA. WASHINGTON,D C,,1979		X											X	X				
U S ENVIRONMENTAL PROTECTION AGENCY CRITERIA AND STANDARDS DIVISION AMBIENT WATER QUALITY CRITERIA FOR POLYCHLORINATED BIPHENYLS EPA-440/5-80-068,WASHINGTON,D C ,OCTOBER 1980	X												X	X				

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	BIO-ASSAYS	FUR-ANS	OTH CHEM	CHAR CHEM	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	REC CHEM	ANAL CHEM	METH CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV (APT	TRNS INFO	TRI/ DISP
VAN MILLER,J P ,J J LALICH,AND J.B ALLEN INCREASED INCIDENCE OF NEOPLASMS IN RATS EXPOSED TO LOW LEVELS OF 2 3,7,8-TETRACHLORODIBENZO-P-DIOXIN CHEMOSPHERE,6:837-844,1977		X												X				
VAN MILLER,J P ,J J LALICH,AND J H ALLEN INCREASED INCIDENCE OF NEOPLASMS IN RATS EXPOSED TO LOW LEVELS OF 2,3,7,8-TETRACHLORODIBENZO-DIOXIN CHEMOSPHERE,6:625-632,1977		X												X				
VANNESS,E F ,ET AL TETRACHLORODIBENZO-P-DIOXINS IN CHEMICAL WASTES, AQUEOUS EFFLUENTS AND SOILS CHEMOSPHERE,9:583-563,1980		X									X							
VECCHI,A ,ET AL COMPARISON OF THE IMMUNOSUPPRESSIVE EFFECT OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) AND 2,3,7,8-TETRACHLORODIBENZO-FURAN (TCDF) IN MICE PRESENTED AT THE DCB DIOXIN SYMPOSIUM,KANSAS CITY, MISSOURI,SEPTEMBER 1982		X	X											X				
VECCHI,A ,ET AL EFFECT OF ACUTE EXPOSURE TO 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN ON HUMORAL ANTIBODY PRODUCTION IN MICE CHEM.BIOL INTERACTIONS, 36:337-342,1980.		X												X				
VEERKAMP,W ,J WEVER,AND B HUTZINGER THE METABOLISM OF SOME CHLORINATED DIBENZOFURANS BY RATS, CHEMOSPHERE 10:397-403 1981,			X									X		X				
VERSAR,INC MICROECONOMIC IMPACTS OF THE PROPOSED PCB BAN REGULATIONS EPA-560/6-77-038,SPRINGFIELD,VA ,MAY 1978	X																X	

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-KINS	FUR-AND	OTH OCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	RFC CHEM	ANAL CHEM	METO CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TOT/BIOP
VOS,J G 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN: EFFECTS AND MECHANISMS IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL,ED ECOL BULL (STOCKHOLM),27:109-176,1978.		X									—	X		X				
VOS J G AND J M KOEMAN COMPARATIVE TOXOLOGIC STUDY WITH POLYCHLORINATED BIPHENYLS IN CHICKENS WITH SPECIAL REFERENCE TO PORPHYRIA, EDEMA FORMATION, LIVER MICROSOMES AND TISSUE DEPOSITIONS TOXICOL APPL.PHARMACOL.,17: 656-668,1970	X		X								X			X				
VOS,J G ,ET AL IDENTIFICATION AND TOXICOLOGICAL EVALUATION OF CHLORINATED DIBENZOFURANS AND CHLORINATED NAPHTHALENES IN TWO COMMERCIAL POLYCHLORINATED BIPHENYLS. FOOD COSMET. TOXICOL .8:625-633,1970	X		X	X							X							
VOS,J G ,J A MOORE,AND J E.ZINKL EFFECT OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN ON THE IMMUNE SYSTEM OF LABORATORY ANIMALS ENVIRON HEALTH PERSPEC .EXPERIMENTAL ISSUE 5:149-163. 1973		X												X				
VOS,J G ,J G KREEFTENBERG,AND L EATON IMMUNE SUPPRESSION BY TCDD IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS F CATTABENI,A CAVALLARO,AND E GALLI EDS SPECTRUM PUBLICATIONS. NEW YORK,1978 PP 163-177		X												X				
WARD C T ,AND F MATSUMURA FATE OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) IN A MODEL AQUATIC ENVIRONMENT ARCH ENVIRON CONTAM TOXICOL ,7: 349-357,1978		X							X										

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MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	METH CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	IRMS INFO	TOI/ DISP
WEITZMAN L .AND L PRUCE. DISPOSING SAFELY OF PCBS: WHAT D AVAILABLE. WHAT D ON THE HAY POWER,125(3): 78-79,1981.	x																	x
WEBING A H ECOLOGICAL CONSIDERATIONS REGARDING MASSIVE ENVIRONMENTAL CONTAMINATION WITH 2,3,7,8-TETRACHLORODIBENZO-PARA-DIOXIN IN: CHLORINATED PHENOXY ACIDS AND THEIR DIOXINS C RAMEL,ED. ECOL.BULL [STOCKHOLM]. 27/289-294,1978.		x							x							x		
WHITESIDE,T THE PENDULUM AND THE TOXIC CLOUD YALE UNIVERSITY PRESS, NEW HAVEN,CONN.,1978.		x											x	x	x	x		
WHITMORE,F C. A FIRST ORDER MASS BALANCE MODEL FOR THE SOURCES, DISTRIBUTION, AND FATE OF PCBS IN THE ENVIRONMENT. EPA-560/6-77-006,VERSAR INC .SPRINGFIELD,VA.,.JULY 1977.	x				x	x			x							x		
WHITMORE,F C DESTRUCTION OF POLYCHLORINATED BIPHENYLS IN DEGREE SLUDGE DURING INCINERATION VERSAR, INC .SPRINGFIELD,VA.,1976	x										x							x
WILLIAMS,D T .AND D J.BLANCHFIELD. SCREENING METHOD FOR THE DETECTION OF CHLORODIBENZO-P-DIOXINS IN THE PRESENCE OF CHLORODIPHENYLS, CHLORONAPHTHALENES, AND CHLORODIBENZOFURANS. J. ASSOC OFF ANAL CHEM .55:83-90,1972		x	x	x							x							
WILSON,D C .AND E T SMITH DEMEDIAL MEASURES FOLLOWING ACCIDENTAL RELEASE OF DIOXIN TO BE PRESENTED AT THE 2D INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS, SALZBURG,AUSTRIA,OCTOBER 1982		x													x			

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFC CHEM	ANAL CHEM	METO CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	TRNS INFO	TRT/DISP
WIPF, H K., ET AL. FIELD TRIALS ON PHOTODEGRADATION OF TCDD ON VEGETATION AFTER SPRAYING WITH VEGETABLE OIL. IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS. F. CATTABENI, A. CAVALLARO, AND G. GALLI, EDS. SPECTRUM PUBLICATIONS, NEW YORK, 1978 PP 201-217.		X				X		X										
WONG, A.S. DETERMINATION OF TCDD IN INDUSTRIAL AND MUNICIPAL WASTEWATERS. EPA-600/4-82-030, CALIFORNIA ANALYTICAL LABORATORY, SACRAMENTO, APRIL 1982.		X									X							
WONG, A S., AND D G. CROSBY. DECONTAMINATION OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) BY PHOTOCHEMICAL ACTION. IN: DIOXIN: TOXICOLOGICAL AND CHEMICAL ASPECTS. F. CATTABENI, A. CAVALLARO, AND G GALLI, EDS. SPECTRUM PUBLICATIONS. NEW YORK, 1978. PP. 185-189		X						X										
WOODYARD, J P., P. NYPHANOWSKI, AND J. TAPPA. PCB EQUIPMENT INVENTORY AND MANAGEMENT PLAN FOR STATE OF CALIFORNIA FACILITIES. PAPER PRESENTED AT THE EPRI PCB SEMINAR, DALLAS, TEXAS, DECEMBER 1981	X																X	
WOOLSON, E A., R F THOMAS, AND P D J ENSOR. SURVEY OF POLYCHLORODIBENZO-P-DIOXIN CONTENT IN SELECTED PESTICIDES. J AGRIC FOOD CHEM., 20:351-354, 1972		X									X							
YAMASAKI, N., T YASUI, AND K MATSUOKA. HYDROTHERMAL DECOMPOSITION OF POLYCHLORINATED BIPHENYLS. ENVIRON SCI. TECHNOL., 14:550-552, 1980	X						X											X

MATRIX OF REFERENCES FOR PCBs

REFERENCE	PCBs	DIO-XINS	FUR-ANS	OTH HCs	CHEM CHAR	B/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	RFC CHEM	ANAL CHEM	METB CHEM	HUM TOX	ANIM TOX	ACCS INCD	ENV IMPT	TANS INFO	TRT/DISP
YANG,G C ,AND A E POHLAND, CATION RADICALS OF CHLORINATED DIBENZO-P-DIOXINS IN: CHLORODIOXINS - ORIGIN AND FATE E H BLAIR,ED ADV CHEM SER , 120:33-43 1973.		x				x												
YOCKIM,R S ,A R ISENSEE,AND G E JONES DISTRIBUTION AND TOXICITY OF TCDD AND 2,4,5-T IN AN AQUATIC MODEL ECOSYSTEM CHEMOSPHERE,7:215-220, 1978		x							x					x		x		
YOSHIHARA,S ,ET AL INDUCTIVE EFFECT ON HEPATIC ENZYMES AND ACUTE TOXICITY OF INDIVIDUAL POLYCHLORINATED DIBENZOFURAN CONGENERS IN RATS, TOXICOL. APPL PHARMACOL ,59:580-588,1981			x									x		x				
YOSHIMURA,T.,AND H IKEDA GROWTH OF SCHOOL CHILDREN WITH POLYCHLORINATED BIPHENYL POISONING OR YUSHO ENVIRON RES ,17:416-425,1978	x		x										x		x			
YOUNG, A L ,C E THALKEN,AND W J CAIRNEY HERBICIDE ORANGE SITE TREATMENT AND ENVIRONMENTAL MONITORING, TR-79-169,U S AIR FORCE OCCUPATIONAL AND ENVIRONMENTAL HEALTH LABORATORY, BROOKS AFB,TEXAS,1979		x		x					x									
YOUNG,A L ENVIRONMENTAL CHEMISTRY AND ENVIRONMENTAL TOXICOLOGY; REVIEW LECTURE TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIOXINS AND RELATED COMPOUNDS, SALZBURG AUSTRIA,OCTOBER 1982		x	x			x			x									

MATRIX OF REFERENCES FOR PCDs

REFERENCE	PCDs	BIO-KINE	FOR-ANS	OTH MCs	CHEM CHAR	O/T CHEM	TEMP CHEM	PHOT CHEM	ENV CHEM	MFG CHEM	ANAL CHEM	DETO CHEM	HUM TOX	ANIM TOX	ACCD INCD	ENV IMPT	IDNT INFO	HIST DISP
YOUNG A L THE CHLORINATED DIBENZO-P-DIOXINS (V): THE SCIENCE OF 2,4,5-T AND ASSOCIATED PHENOXY HERBICIDES G W GOVEY AND A L YOUNG,EDS WILEY, NEW YORK,1981 PP 133-208		X			X				X	X			X	X	X	X		
YOUNG,A L ,ET AL THE TOXICOLOGY,ENVIRONMENTAL FATE AND HUMAN RISK OF HERBICIDE ORANGE AND ITS ASSOCIATED DIOXIN TR-79-92,U S AIR FORCE OCCUPATIONAL AND ENVIRONMENTAL HEALTH LABORATORY,BROOKS AFB,TEXAS,OCTOBER 1979		X							X				X	X	X			
YOUNG,A L ,W J GAIBNEY,AND D.CHAMBELL PERSISTENCE, MOVEMENT AND RECONTAMINATIN STUDIES OF TCDD IN STORAGE SITES HAVING BEEN CONTAMINATED WITH PHENOXY HERBICIDES TO BE PRESENTED AT THE 3RD INTERNATIONAL SYMPOSIUM ON CHLORINATED DIBENZO AND RELATED COMPOUNDS,BALTIMORE,AUGUST14,OCTOBER 1983.		X							X									
ZACK,J A ,AND R R SUSKIND THE MORTALITY EXPERIENCE OF WORKERS EXPOSED TO TETRACHLORODIBENZODIOXIN IN A TRICHLOROPHENOL PROCESS ACCIDENT JOH, J OCCUP MED.,22:11-14,1980.		X											X		X			
ZITKO V POLYCHLORINATED BIPHENYLS AND ORGANOCHLORINE PESTICIDES IN SOME FRESHWATER AND MARINE FISHES, BULL ENVIRON CONTAMIN TOXICOL,6:464-470,1971	X			X							X							
ZITKO,V ,D HUTZINGER,AND P M K CHOI CONTAMINATION OF THE BAY OF FUNDY-GULF OF MAINE AREA WITH POLYCHLORINATED BIPHENYLS, POLYCHLORINATED TERPHENYLS, CHLORINATED DIBENZODIOXINS, AND DIBENZOFURANS. ENVIRON.HEALTH PERSPEC ,EXPERIMENTAL ISSUE 1:47-60, 1972	X	X	X						X		X							

Legend

PCBs	- Polychlorinated biphenyls
DIOXINS	- Polychlorinated dibenzo-p-dioxins
FURANS	- Polychlorinated dibenzofurans
OTH HCS	- Other hydrocarbons
CHEM CHAR	- General chemical characteristics
D/T CHEM	- Decomposition/transformation chemistry
TEMP CHEM	- High-temperature chemistry
PHOT CHEM	- Photochemistry
ENV CHEM	- Environmental chemistry
MFG CHEM	- Manufacturing chemistry
ANAL CHEM	- Analytical chemistry
METB CHEM	- Metabolic chemistry
HUM TOX	- Human toxicity
ANIM TOX	- Animal toxicity
ACCD INCD	- Accidents and incidents
ENV IMPT	- Environmental impacts
TRNS INFO	- Transformer and capacitor information
TRT/DISP	- Treatment and disposal

EPRI CS-3308

Below are five index cards that allow for filing according to the four cross-references in addition to the title of the report. A brief abstract describing the major subject area covered in the report is included on each card.

EPRI CS-3308
RP1283-11
Final Report
November 1983

EPRI

State-of-the-Art Review: PCDDs and PCDFs in Utility PCB Fluid

Contractor: SCS Engineers, Inc.

Highly toxic chlorinated aromatic hydrocarbons have been associated with PCBs used in some electrical transmission and distribution equipment. This first large-scale systematic review of available information should help utilities separate fact from conjecture about these substances and their formation. 228 pp.

EPRI Project Manager: R. Y. Kemel

Cross References:
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2. RP1283-11
3. Heat, Waste, and Water Management Program
4. PCBs

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