Monsanto

FROM (NAME—LOCATION—PHONE): H. C. Godt, Jr. - 04B (4-2616)

DATE: May 24, 1984

cc:

SUBJECT: SPECIAL REPORT ON NON-INDUSTRIAL SOURCES OF CHLORINATED DEBENZO-P-DIOXINS (CDDs) - REPORT NO. MSL - 3506

REFERENCE:

TO:
R. J. Barnes - Townley & Updike
T. M. Bistline - E2ND
A. M. Ford - G3WG
W. J. McCarville - G3WG
P. M. Pleska - Bowles, McDavid, Graff & Love
G. Roush - G2WG
D. F. Snively - E2ND
M. S. Weinberg - Weinberg Consulting Group, Inc.
J. D. Wilson - G3WG
Litigation Technical Support Archives (A. M. Ford) (2 copies)
Technical Reports Library (R. G. Lee - R2C) (2 copies)

A month has elapsed since the subject report was issued. In the meantime, Allan Ford, Jim Wilson and I have carefully perused the report looking for errors and misrepresentations of all kinds. Attached are the following corrected pages:

Volume 1:

2, 4, 5, 6, 7, 9, 13, 14, 15, 16, 18, 19, 21, 22, 23, 26, 27, 35, 37, 39, 40, 41, 43, 47, 49, 50, 51, 53, 54, 56, 58, 59, 60, 69, 70, 71, 73, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 92, 96, 97, 99, 103, 104, 109, 110, 112, 122, 125, 128.

Volume 2:

15, 229, 230.

Please replace the appropriate pages in your copy of the report. If you have any questions, please contact me.

H. C. Godt, Jr.

H. C. Godt, Jr.

/mll
Attachments

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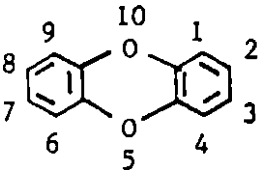
SUBJECT TO PROTECTIVE ORDER.

Table 1
Chlorinated Dibenzo-p-dioxin Congeners[a] by Homolog[b] and Isomer[c]



Monochloro (2 isomers)
1-;2-

Dichloro (10 isomers)
1,2-; 1,3-; 1,4-;
1,6-; 1,7-; 1,8-;
1,9-; 2,3-; 2,7-;
2,8-.

Trichloro (14 isomers)
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 isomers)
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 isomers)
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-;
1,2,3,7,9-; 1,2,3,8,9-;
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 isomers)
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 isomers)
1,2,3,4,6,7,8-;
1,2,3,4,6,7,9-.

Octachloro (1 isomer)
1,2,3,4,6,7,8,9-.

Molecular Weights of Homologs

Monochloro	=	218.64
Dichloro	=	253.08
Trichloro	=	287.52
Tetrachloro	=	321.96
Pentachloro	=	356.40
Hexachloro	=	390.84
Heptachloro	=	425.28
Octachloro	=	459.72

a. Congener = a general term used to mean any isomer of any homolog; thus there are 75 PCDD congeners.

b. Homolog (isomer cluster) = a group of PCDD isomers having a specified number of chlorine atoms; thus the tetrachloro homolog has 22 isomers.

c. Isomer = a PCDD with a specific numerical arrangement of the chlorine atoms within a homolog.

One of the striking features of 2,3,7,8-TCDD's toxicity in animals is that the lethal dose varies so much from one species to another. Table 4 illustrates this fact.

Table 4
Variation of 2,3,7,8-TCDD Toxicity
in Different Species

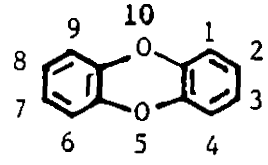
Animal	LD_{50}(ug per kg body weight)
Guinea pig	1
Rat(male)	22
Rat(female)	45
Monkey	<70
Rabbit	115
Mouse	114
Dog	>300
Bullfrog	>500
Hamster	5000

Source: Poland and Knutson, Annual Review of Pharmacology & Toxicology, 1982

The guinea pig, the most sensitive animal tested, is at least 5000 times more sensitive than the hamster, the least sensitive animal yet tested. Rabbits, mice, and monkeys are somewhere in the middle - roughly 100 times less sensitive than guinea pigs, but 50 times more sensitive than hamsters.

A. Structure-Biological Activity Relationship

It is important to note the structure-activity relationship among the CDDs. The unusual toxicity of the chlorinated dibenzo-p-dioxin molecule is associated with the positioning of chlorine on the planar, tricyclic ring system. As early as 1973, Kende and Wade [111] analyzed the structure-activity relationship of about two dozen PCDD congeners. Recently, Poland [79] and Payne [117] noted certain other characteristics. The following presents the structure-activity relationship as now conceived:



1. halogen atoms must occupy at least three, and for maximum potency, four of the lateral ring positions (positions 2,3,7 and 8)

2. at least one ring position must be unsubstituted

3. no amount of halogen substitution on only one ring leads to biological activity.

4. halogenation in the peri positions (positions 1,4,6 and 9) reduces potency; however, reduction in potency due to peri position halogenation is not nearly as significant as the removal of halogen from one or more of the lateral positions.

5. the order of potency with regard to halogen substitution is bromine >chlorine >fluorine (potency of iodine analog apparently not determined).

6. other functional groups [e.g., nitro ($-NO_2$)] render the dibenzo-p-dioxin ring toxic, but the potency is considerably less.

7. 2,3,7,8-TCDD and its approximate congeners can be thought of as roughly fitting into a rectangle 3 x 10Å (angstroms) with chlorine atoms in the four corners.

It has been suggested that the extreme toxicity of 2,3,7,8-TCDD and the specific penta- and hexachloro isomers mentioned is related to the detoxification process of hydroxylation which takes place exclusively at the lateral positions. The presence of the chlorine atoms at these positions may prolong the in vivo life of the laterally substituted PCDDs, and thereby increase the toxicity of much smaller doses [117].

Industrial processes that produce CDDs as unwanted by-products generally react specific materials to produce specific products. Thus, specific CDD congeners are found among the reaction products. This is not true of most of the non-industrial sources, which produce a much broader distribution of both toxic and relatively non-toxic CDDs. The non-industrial sources generally produce measureable quantities of 2,3,7,8-TCDD but it is not one of the major isomers present. The importance of the non-industrial sources of CDDs is related to the mass transport rate of the toxic CDD isomers and analogs (e.g., 2,3,7,8-TCDF) rather than the proportionality of the individual components.

Because of the broad distribution of the toxic CDDs present in the solid and gaseous effluents from combustion where 2,3,7,8-TCDD represents a very small part of the total quantity of toxic PCDD isomers and PCDF analogs present, attempts have been made to estimate the "toxic equivalents" of the PCDDs and PCDFs emitted relative to 2,3,7,8-TCDD. One such study is that of Olie, Lustenhouwer and Hutzinger, who calculated the "toxic equivalents" in the fly ash and flue gases from municipal incinerators in the Netherlands. [76] Since the toxicity of nine of the 19 probable toxic PCDD isomers and only eight of the 39 probable toxic PCDFs have been studied, an assumption was made that the other probable toxic PCDD and PCDF isomers have a toxicity roughly 10% of that of

2,3,7,8-TCDD. From the concentrations of the isomer clusters of PCDDs and PCDFs with the same number of chlorine atoms (TCDDs, PeCDDs, HPDFs, etc.) determined in the fly ash and flue gas extracts and knowing that 2,3,7,8-TCDD is about 3.3% of the tetra isomers, the "toxic equivalents" relative to 2,3,7,8-TCDD were calculated. Table 5 gives the results.

Table 5
"Toxic Equivalents"(as 2,3,7,8-TCDD) in Emissions from Municipal Incinerators in the Netherlands

	Fly Ash			Flue Gas		
Compounds	Total Amt./Yr. (kg)	Toxic Equiv./Yr. (kg)	2,3,7,8-TCDD (kg)	Total Amt./Yr. (kg)	Toxic Equiv./Yr. (g)	2,3,7,8-TCDD (g)
TCDDs	5.6	0.3	0.2	0.8	41	26
PeCDDs	15.2	0.8		3.4	170	
HCDDs	36.2	2.5		6.2	434	
HpCDDs	45.6	2.3		4.9	245	
TCDFs	10.4	0.2		2.3	48	
PeCDFs	18.7	0.9		3.8	190	
HCDFs	27.5	2.1		7.4	555	
HpCDFs	18.8	0.9		4.1	205	
Total	178.0	10.0		32.9	1888	

Assumptions: 2,3,7,8-TCDD = 3.3% of total TCDDs.
All "toxic" compounds have a toxicity of 10% compared to 2,3,7,8-TCDD.
Isomers are equally distributed in each isomer group.

Based on the above results, the authors estimate that the toxicity of the fly ash extracts is about 50 times greater than that expected from the 2,3,7,8-TCDD content. For the flue gas, the factor is about 80. It is very obvious that the penta, hexa, and hepta CDDs and CDFs contribute greatly to the toxicity of the emission extracts, and 2,3,7,8-TCDD probably contributes very little. An article by Helder, [36] published in 1982, confirms this estimate. Fly ash extract was extremely toxic to rainbow trout yolk sac fry, which was attributable for the greater part to the other chorinated PCDD congeners and only a small part to 2,3,7,8-TCDD.

B. Histopathology and Cause of Death

The toxicological and histopathic response to 2,3,7,8-TCDD and its congeners include [79]:

1. Lethality - The ultimate target organ, that tissue whose functional disruption leads to death, is unknown. Animals show a slow wasting syndrome with a latent period before death.

2. Lymphoid involution - produces thymus, lymph node, and spleen atrophy accompanied by suppression of cellular immunity.

3. Embryotoxicity and teratogenesis - it is a potent embryotoxin and teratogen in mice, rats and chicken embryo.

4. Chloracne and hyperkeratosis - most common and characteristic sign of toxicity in humans. Also observed in rabbits, monkeys and hairless mice.

5. Liver damage - heptacellular necrosis observed in rats, mice and rabbits. Also, a disturbance in hepatic porphyrin synthesis (hepatic porphyria).

6. Edematous syndrome - a characteristic lesion in chickens. Also seen in mice.

7. Bone marrow depression - reported in monkeys.

8. Carcinogenesis [118] - demonstrated in rats treated with 0.1 ug/kg body weight of 2,3,7,8-TCDD for two years, but no adverse effects at 0.001 ug/kg body weight. NIOSH and EPA water quality criteria currently list it as a carcinogen. Carcinogenesis in humans is indefinite.

There is evidence that 2,3,7,8-TCDD is slowly biologically attacked yielding metabolites appearing in the bile. There appears to be no reports of toxicity on any cell type culture.

Most significant is the fact that 2,3,7,8-TCDD is an excellent enzyme inducer, and produces a 10 to 12 fold increase in hepatic aryl hydrocarbon hydroxylase (AHH) and δ-aminolevulinic acid synthetase (ALAS) activity. This is very important because there is an excellent correlation between the potency of the PCDDs and their congeners to induce AHH and ALAS activity and their toxicity. A comparison of the enzyme inducing efficiency of various PCDDs is given in Table 6 [117].

Table 6
Induction of ALAS and AHH by PCDDs in Liver of Chick Embryos

Chlorodioxin	ALAS	AHH
Unsubstituted	–	–
Mono I	–	–
Di 2,3	–	–
2,7	–	–
2,8	–	–
Tri 1,2,4	–	–
2,3,7	+ +	+ +
Tetra 1,2,3,4	–	–
1,3,6,8	–	–
2,3,7,8	+ + + +	+ + + +
Penta 1,2,3,4,7	+ +	+ +
Hexa 1,2,3,4,7,8	+ +	+ + + +
1,2,4,6,7,9 (90 per cent)	±	+
Octa 1,2,3,4,6,7,8,9	–	–

III. NON-INDUSTRIAL SOURCES

Until about six years ago, most of the attention given to the sources of CDDs centered around their production as impurities in industrial processes. This was brought about primarily because of certain accidents which occurred such as the "run-away" reaction at Monsanto Company's Nitro plant in 1949 in the production of sodium 2,4,5-trichlorophenate (NaTCP), an intermediate in the production of 2,4,5-T, and a similar, more violent incident in Seveso, Italy in 1976.

In 1977, Olie and co-workers [73] reported the finding of CDDs as trace components in the fly ash and flue gas from some municipal incinerators in the Netherlands. This was the first reported case of the observance of CDDs from a non-industrial source. Since then, numerous studies from industrial, academic and government laboratories all over the world have reported finding CDDs and the closely related chlorinated dibenzofurans (CDFs) in many different forms of combustion - some being adequately confirmed and others remaining controversial. To the best of our knowledge, the only non-industrial sources of CDDs reported to date involve combustion in one form or another. The possibility of CDDs being formed in biological environmental processes has been suggested [54], but no reports of such findings have been documented.

In this section, we will report the findings of the researchers regarding the various combustion sources where CDDs have been identified. The major combustion sources implicated will be discussed individually and chronologically. Laboratory studies of the combustion or thermal destruction of chemical compounds to determine the mechanism of formation of CDDs will be covered in the Chemistry section of this report. Laboratory studies directly related to non-industrial sources of CDDs will be covered under the specific source.

A. Municipal Incinerators

The first report citing CDDs from a non-industrial source was by Olie [73] and co-workers in 1977. They collected fly ash from the electrostatic precipitator (ESP), and particulates and liquid condensate from flue gas from three municipal incinerators in Arnheim, Amsterdam, and Alkmaar, Holland. The incinerators were modern, large and representative installations geographically distributed in different areas of Holland. Chemical waste, as a rule, was not incinerated. The emission samples were extracted with appropriate organic solvents. After removing most of the solvent, the extracts were examined by packed column gas chromatography/ mass spectrometry (GC/MS) without further purification. In the fly ash were found tetrachloro-, pentachloro-, hexachloro-, heptachlorodibenzo-p-dioxin isomers (TCDDs,

Table 11
PCDDs and PCDFs in Ash Samples of the Zurich-Hagenholtz Trash Incinerator (in ppb)*

	Ash 1 (furnace) 900°C.	Ash 2 (behind superheater) 480°C.	Ash 3 (in front of economizer) 480°C.	Ash 4 (behind electrofilter) 260°C.
OCDD	6	18	n.d.	120
HpCDDs	n.d.	n.d.	n.d.	60
HCDDs	n.d.	n.d.	n.d.	30
PeCDDs	n.d.	n.d.	n.d.	8
TCDDs	n.d.	n.d.	n.d.	2
OCDF	n.d.	15	n.d.	10
HpCDFs	n.d.	12	n.d.	40
HCDFs	n.d.	1	n.d.	30
PeCDFs	n.d.	n.d.	n.d.	4
TCDFs	n.d.	n.d.	n.d.	1

* Samples of Oct. 5, 1977
n.d. = not detectable. Detection limits: 0.2 (TCDDs/TCDFs) to 1 ppb (OCDD/OCDF).

From Table 11, the largest concentration of PCDDs and PCDFs is in the fly ash from behind the elctrofilter, the last attainable spot before the chimney. It is also the coolest sample point. Ash samples 1 to 3, taken at considerably higher temperatures, contained only the most chlorinated (least volatile) PCDDs and PCDFs.

Considerable quantities of chlorobenzenes (PCBzs), chlorinate biphenyls (PCBs) and chlorinated naphthalenes (PCNs) were also found. The authors indicated that they did not know if the PCDDs were present in the waste and passed through the incinerator unchanged or were formed in the incinerator from chlorinated aromatic precursors. They suggested that the latter was most likely.

Another publication in 1978 by the authors together with C. Rappe[80] gives the identification of many of the PCDD isomers found in the Zurich-Hagenholtz incinerator ash by sharpening up the GC/MS analysis by using the SIM technique with standards. Although the data was not quantitative, 12 of the possible 22 TCDD isomers were present. The main isomers were 1,3,6,8- and 1,3,7,9-TCDD. A small amount of 2,3,7,8-TCDD was identified. Up to 10 of the possible 14 PeCDD isomers were found with 1,2,3,7,8- and 1,2,4,7,8-PeCDD being identified. Up to 8 of the possible 10 HCDD isomers were present, the main ones being 1,2,3,4,6,8-, 1,2,3,6,8,9-, 1,2,3,6,7,8- and 1,2,3,7,8,9-HCDD. Both possible HpCDD isomers (1,2,3,4,6,8,9- and 1,2,3,4,6,7,8-HpCDD) and OCDD were also present. Most important was the fact that micropyrolysis of a mixture of the potassium salts of 2,4,6-tri, 2,3,4,6-tetra and pentachlorophenol gave the same pattern of main isomers identified in the fly ash from the Zurich-Hagenholtz incinerator as well as from an industrial waste incinerator. This was the first concrete evidence that chlorophenols could be precursors of PCDDs in combustion. In the Chemistry section, we will elaborate on this point.

In November, 1978, Dow issued a report by its Chlorinated Dioxin Task Force to the Michigan Dept. of Natural Resources, which indicated that PCDDs are found in widely differing incineration processes including municipal incinerators. This report first mentioned their "Trace Chemistries of Fire" hypothesis, which has led to much controversy. We do not have a copy of this original report, but the technical data appeared in an article by Bumb, et al, in Science in 1980 [13]. Dow's "Trace Chemistries of Fire" hypothesis says "Chlorinated dioxins appear to be ubiquitous. Their ubiquity is due to the existence of natural phenomena, trace chemistries of fire, which consist of numerous chemical reactions occurring during combustion of concentrations as low as 10^{-10} percent." Dow supports their case for the "Trace Chemistries of Fire" hypothesis by the data presented in Table 12, which shows that PCDDs are emitted from various combustion sources, as well as being found in soil, dust, and wastewater sludge (Milorganite) samples.

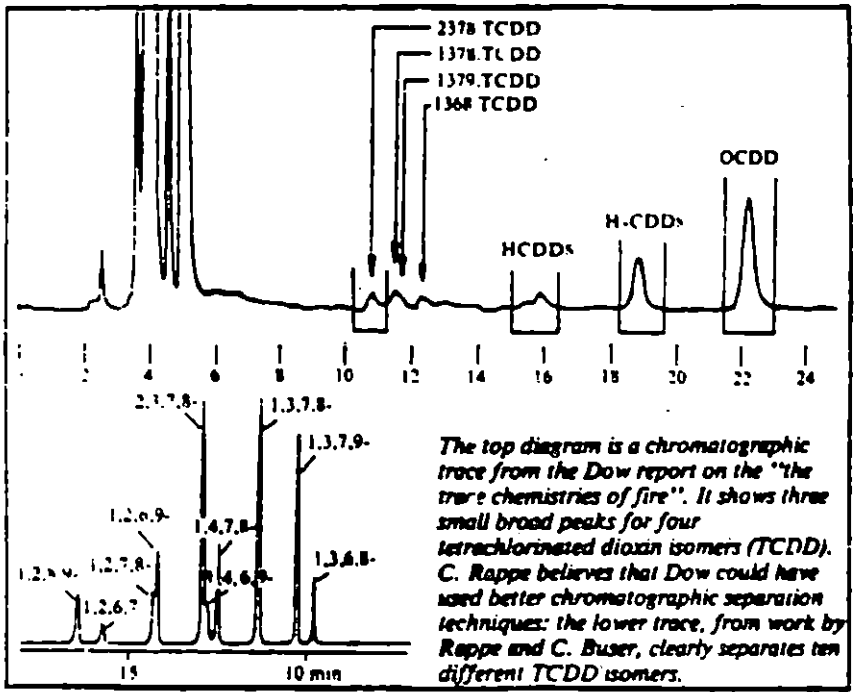
Table 12
Chlorinated Dioxins in the Environment

Sample	Number of samples	Apparent dioxin content, ng/g (ppb)*					
		TCDD			HCDD	H_7CDD	OCDD
		Other isomers	2,3,7,8-	Total			
Soil							
Rural (Gaylord, Michigan)	5			N.D.†	N.D.	N.D.-0.05	N.D.-0.2
Urban (Lansing and East Lansing, Michigan)	5			N.D.	0.03-1.2	0.03-2	0.05-2
Major metropolitan (Chicago, Illinois)	8			0.005-0.03	0.03-0.3	0.1-3	0.4-22
Dow Chemical (Midland, Michigan)	5	0.8-18	0.3-100	1-120	7-280	70-3,200	490-20,000
Dust							
Dow Chemical laboratory	6	0.5-2	0.7-3	1-4	9-35	140-1,200	650-7,500
Midland, Michigan	2			0.03-0.04 (0.02)	0.2-0.4	2-4	20-30
Metropolitan (Detroit, Michigan)	4			N.D.-0.03	N.D.-0.3	0.3-4	0.1-4
Metropolitan (St. Louis, Missouri)	1	0.16	0.12	0.3	2	34	210
Metropolitan (Chicago, Illinois)	2			0.04 (0.04)	N.D.-0.3	0.6-3	3-8
Wastewater treatment sludge							
Commercial sludge fertilizer (Milwaukee, Wisconsin)	1	0.29	0.02	0.31	2	30	180
Incinerators, powerhouse							
Dow powerhouse	1	38 (20)	N.D. (10)	38 (20)	2	4	24
Dow rotary incinerator stack (present normal operation with supplemental fuel)‡	5			N.D. (2)	1-5	4-100	9-950
Dow stationary tar burner stack (normal operation with supplemental fuel)	5	N.D.	N.D.	N.D.	1-20	27-160	190-440
U.S. municipal incinerator (electrostatic precipitator) (Nashville, Tennessee)	1	7.3	0.4	7.7	14	28	30
European municipal incinerators				2-20	30-200	60-130	40-120
Mufflers							
Diesel truck muffler	2	0.02	0.003	0.023	0.020	0.100	0.26
Auto muffler	4	N.D.-0.004	N.D.-0.004	N.D.-0.008	N.D.	0.003-0.01	0.02-0.07
Other sources							
Home fireplace soot	2	N.D.-0.3	N.D.-0.1	N.D.-0.4	0.2-3	0.7-16	0.9-25
Home electrostatic precipitator	1	0.4 (0.4)	0.6	1.0	34	430	1,300
Cigarette smoke	2	N.D.	N.D.	N.D.	0.004-0.008	0.009	0.02-0.05
Charcoal-broiled steak	4	N.D.	N.D.	N.D.	N.D.	N.D.	0.03 (0.03)

*Where multiple samples were analyzed from similar sources, the range of observed values is shown. †N.D. indicates that the signal observed was less than 2.5 times noise. Limits of detection generally observed were TCDD, 0.001 to 0.01 ppb; HCDD, 0.01 to 0.05 ppb; H_7CDD, 0.003 to 0.03 ppb; and OCDD, 0.01 to 0.05 ppb. Limits of detection not within these ranges are shown parenthetically after the value reported for signals between 2.5 and 10 times noise. ‡Data without supplemental fuel not included here because this practice has been eliminated.

Note that Dow examined the fly ash from a municipal incinerator in Nashville, Tenn. and found PCDDs. Also approximate data for European municipal incinerators are shown for comparison. Notice that 2,3,7,8-TCDD is only a small part of the TCDDs present in the fly ash from the Nashville incinerator. The analytical methodology including sampling was reviewed by a group of scientists, who found it to represent state-of-the-art. However, in October, 1979, A. Hay [35] reported that C. Rappe was severely critical of the Dow study after reading the original November, 1978 report. He questioned the methodology, particularly the chromatographic separation techniques used by Dow to identify PCDD isomers. He said Dow's techniques were poor (see Figure 1), leading to an over estimation of the amount of 2,3,7,8-TCDD present in the fly ash

Figure 1



The top diagram is a chromatographic trace from the Dow report on the "the true chemistries of fire". It shows three small broad peaks for four tetrachlorinated dioxin isomers (TCDD). C. Rappe believes that Dow could have used better chromatographic separation techniques: the lower trace, from work by Rappe and C. Buser, clearly separates ten different TCDD isomers.

from municipal incinerators. Rappe also indicated that the ubiquitous nature of PCDDs was far from proven. At that time, Rappe considered chlorinated phenols and diphenyl ethers to be the main precursors to PCDDs in municipal incinerator emissions, based on laboratory combustion experiments on these two classes of compounds giving PCDD isomers in similar proportions to those found in commercial incinerators. He said he reported these findings to Dow, who apparently ignored them because there are no analyses for these apparent precursors in the Dow report.

In 1982, an article by Crummett [23] reviews much of the data presented by Bumb, et al, [13] and elaborates on the analytical methodology. The analytical procedures employed several liquid chromatographic (LC) cleanup processes and various column GC/low and high resolution MS techniques. Crummett reemphasizes the apparent ubiquitous nature of PCDDs and cites Mahle and Whiting's study [67] on the pyrolysis of essentially chlorinated dioxin-free coal in the presence of sodium chloride, hydrogen chloride and chlorine giving rise to PCDDs. He says these studies support the "Trace Chemistries of Fire" hypothesis and the fact that PCDDs appear to be ubiquitous is reasonable.

Table 13

Chlorinated Dioxins From Thermal Oxidation and Chlorination of Coal

Material Heated	Apparent Chlorinated Dioxins, ng/g (ppb)			
	TCDD	HCDD	H_7CDD	OCDD
Cl_2 + Air	ND(0.07)	ND(0.2)	ND(0.3)	ND(0.5)
Coal + Air	ND(0.08)	ND(0.1)	0.6(0.3)	1.3(0.7)
Coal + Air + NaCl	ND(0.07)	ND(0.2)	0.5(0.2)	2.7(0.5)
Coal + Air + HCl	1.4(0.006)	8	25	64
Coal + Air + Cl_2	1.2	29.	91.	290.

In 1979, Eiceman, Clement, and Karasek [20] analyzed fly ash samples from municipal incinerators in Canada, Japan and the Netherlands. The Canadian samples were from two city incinerators, one located in a heavily industrialized area and the other located in a more domestic region of the city. The Netherlands sample was from O. Hutzinger at the Univ. of Amsterdam. The five samples were extracted with benzene, and the concentrated extracts examined by GC/MS using the SIM technique. PCDDs and PCDFs (Cl_4 through Cl_8 isomers) were present in all samples. This suggested to the researchers that PCDDs and PCDFs are formed universally during incineration although the combustible materials and conditions may vary.

In 1981 and 1982, these researchers extended their studies of PCDDs from Canadian municipal incinerators [19,21]. Eight samples of fly ash were taken weekly from a single municipal incinerator in Ontario, Canada. Table 14 shows the results using GC/MS analysis with SIM technique. The values ranged from 0.4ng/g for OCDD in sample 7 to 53ng/g for HCDDs in sample 3. The largest change in concentration of a chlorine number isomer cluster occurred in the HCDDs where samples 3 and 7 differed by 51ng/g. PeCDDs and HCDDs were present in the highest concentrations. PAHs were also measured.

Table 15
Representative Quantitative Results of Analyses of PCDDs and PCDFs(ppb) in Solid Emissions from 25 Municipal Incinerators

Isomer Groups	unpublished[a] Solid Emissions			
	fly ash[a] (electr. prec.)			part. matter (stack)
TCDDs	5	54	110	100
PeCDDS	31	182	488	800
HCDDs	80	326	1200	1370
HpCDDs	190	288	902	1370
OCDD	266	106	110	310
TCDFs	13	111	223	460
PeCDFs	42	196	510	960
HCDFs	109	361	870	1600
HpCDFs	89	177	407	1130
OCDF	20	18	26	140

a. low, medium and high values chosen from 80 analyses (over 25 installations studied)

In 1980 and 1981 [16,83] A. Cavallaro studied the emissions from various sources including municipal incinerators in the Lombardy Region of Italy. The slag, fly ash and liquid condensate samples were extracted and the extracts purified by layered column liquid chromatography and analyzed for PCDDs and PCDFs by HRGC/LRMS with EI or MNCI source and SIM technique with sensitivity in the pg (ppt) range. One of the objects of these studies was to determine if PCDDs and PCDFs were present in the emissions of all types of incinerators fed with different type wastes. Every examined sample from the different incinerators exhibited a similar profile concerning the presence of PCDDs and PCDFs, a variable number of isomers from TCDDs to OCDD and from TrCDFs to OCDF being detected, with many specific isomers identified. Generally the hexa, hepta, and octa CDDs and CDFs predominate over the penta and tetrachloro isomers. This has significance because degradation proceeds by dechlorination rather than ring cleavage, thus the higher chlorinated species (higher than tetra) degrade through the tetrachloro isomers. Table 16 gives the analytical results from 6 municipal solid waste incinerators. Notice that in nearly all cases the vapor phase (condensate) contains considerably larger amounts of PCDDs and PCDFs than does the dusts (solid phase). Many researchers have only looked at fly ash because sampling is easier.

Table 16
Analyses of Emissions from Six Municipal
Incinerators in Lombardy Region (Milan) of Italy

Installation	Type of sample	Σ 4-CDD	Σ 5-CDD	Σ 6-CDD	Σ 7-CDD	8-CDD	Σ 4-CDF	8-CDF
n. 1	Dusts from dust traps ppb	*	*	*	*	*	*	*
	Emitted dusts ng/Nm^{3}U	1,1	2,7	11,5	1,03	8,0	*	2,2
	Emitted condensate ng/Nm^{3}U	19,6	27,9	178,2	159,6	63,9	*	59,3
n. 2	Dusts from dust traps ppb	0,25	1,7	294,0	8,9	295,0	0,46	15,8
	Emitted dusts ng/Nm^{3}U	172,2	172,3	12015,0	575,0	7312,0	75,0	2883,0
	Emitted condensate ng/Nm^{3}U	17,0	107,0	26620,0	828,0	1179,0	108,6	4390,0
n. 3	Dusts from dust traps ppb	N.D.	0,92	1,8	3,1	1,5	0,8	3,3
	Emitted dusts ng/Nm^{3}U	0,037	0,3	6,7	0,2	1,7	2,57	0,08
	Emitted condensate ng/Nm^{3}U	19,0	40,0	6542,0	124,0	776,0	429,0	1010,0
n. 4	Dusts from dust traps ppb	46,4	65,4	2496,0	87,9	841,5	61,7	255,0
	Emitted dusts ng/Nm^{3}U	10,9	2,8	0,54	3,2	39,0	3,7	0,06
	Emitted condensate ng/Nm^{3}U	60,0	33,0	1390,0	167,0	2703,0	1814,0	1760,0
n. 5	Dusts from dust traps ppb	0,7	0,05	0,021	0,007	0,1	1,18	0,0015
	Emitted dusts ng/Nm^{3}U	0,34	2,4	196,0	9,9	173,0	75,3	3,2
	Emitted condensate ng/Nm^{3}U	9,6	21,0	328,0	46,0	244,0	305,0	89,0
n. 6	Dusts from dust traps ppb	N.D.	N.D.	N.D.	0,0012	5,86	N.D.	1,93
	Emitted dusts ng/Nm^{3}U	N.D.	0,010	0,28	N.D.	0,51	N.D.	N.D.
	Emitted condensate ng/Nm^{3}U	19,0	11,0	480,0	6,0	71,0	27,0	24,0

* = not investigated
N.D. = not determined

From 1980 to 1982, Liberti, Brocco and co-workers [11,58,59,60,61,62] also investigated the emissions from municipal incinerators in Italy. The emissions included bottom ash (from furnace), fly ash (from ESP), sludge (fly ash collected by water spraying the fumes), particulates (from stack fumes) and organic vapors (condensed from stack fumes). The extracts of the samples were cleaned up by use of multilayered column liquid chromatography and analysis was by capillary HRGC/MS with EI source and MID technique. In one of their publications [58], they analysed the starting refuse to be incinerated and found no PCDDs or PCDFs. This is one of the few reports which clearly shows that the PCDDs and PCDFs are formed "by pyrolytic reactions of precursors". These investigators support Dow's "Trace Chemistries of Fire" hypothesis, and seem to concur that PCDDs are ubiquitous. They say "Our investigations show that the incineration processes of urban wastes definitely yield a number of PCDDs and PCDFs through a variety of possible reactions, the concentration being low definitely but much larger than those usually found in the environment and arising from other sources."

Table 19 [62] gives more specific analyses of emissions of an urban Italian incinerator. The dry wastes are a sample of the wastes which were incinerated. Notice that no PCDDs were found; however, PCBs and chlorophenols were found. Also the higher the PCBs in the emission sample, the higher were the PCDDs in the same sample. Likewise, the highly chlorinated PCDDs are present in the largest concentration with OCDD being the most.

Table 19
Concentration of Some Chloroorganic Compounds
in the Emissions of an Urban Italian Incinerator

Peak No.	Compounds	Dry wastes (ng/g)	Ash (ng/g)	Fly ash (ng/g)	Fume part. matter (ng/g)
	PCB_{TOT}	200	550	1,100	7,800
13	2,3,7,8- and/or 1,2,3,4-tetra-CDD	-	trac.	trac.	40
16	not id.	-	2	9	115
17	penta-CDD	-	4	6	110
20	not id.	-	4	7	160
21	1,2,4,6,7,9-hexa-CDD	-	2	7	150
22	not id.	-	6	24	400
23	hexa-CDD	-	4	15	250
24	1,2,3,6,7,8-hexa-CDD	-	4	7	140
26	not id.	-	15	210	-
27	hepta-CDD	-	8	25	400
28	hepta-CDD	-	8	30	500
32	octa-CDD	-	35	50	900

In another publication by Liberti, Brocco and co-workers [60], various emissions from Italian urban waste incinerators classified as follows were analysed:

A. incinerators from highly populated areas where wastes are incinerated without any treatment.

B. incinerators where waste is mainly agricultural products which are burned without any treatment.

C. incinerators where waste passes through a recycling process where paper, plastics and vegetable material are removed.

D. incinerators where the input material is the residue of wastes which went through an homogenization and oxidation process (the unfermented residue being burned).

The various emission samples were extracted, and the extracts purified by passage through silica gel and alumina chromatographic columns. Analyses were performed by packed or capillary column GC/MS with EI source and SIM technique. Table 20 gives the analytical

Table 20
Concentration (ppb) of PCDDs and PCDFs in Emissions from Italian Municipal Incinerators Burning Wastes of Different Types

Incinerator	Emission analysed	PCDD n. Cl=4	5	6	7	8	PCDF n. Cl=7	8
A	fly ash	20	100	160	230	490	70	50
A	ash	16	20	33	90	340	25	50
A	Particulated in fumes	n.e	80	180	290	510	110	60
B	fly ash	n.e	n.e	n.e	30	40	n.e	n.e
B	sludge	n.e	n.e	n.e	10	15	n.e	n.e
C	sludge	n.e	n.e	n.e	6	12	n.e	n.e
D	Particulated in fumes	n.e	n.e	n.e	5	5	n.e	n.e
D	sludge	n.e	n.e	n.e	1	1	n.e	n.e

n.e. = not evaluable

results. These researchers propose the following simple mechanism of formation of PCDDs and PCDFs:

Precursor A (Organic matrix): Phenols, Polyphenols, Materials with phenolic structure
+
Precursor B (Cl_2 or HCl donor): PVC, Other chlorinated plastics, Chlorinated pesticides
$\longrightarrow$ PCDDs + PCDFs

Examples of Precursor A are tannins widely diffused in vegetables, flowers and fruits which upon heating yield phenols and polyhydroxyphenols. This simple hypothesis explains the analytical results observed from the various types of incinerators. Type B incinerators give lower concentrations of PCDDs and PCDFs than Type A incinerators. Vegetable matter will have only small amounts of precursor B, thus only small amounts of chlorinated compounds will appear in the emissions. For type C and D incinerators, most A precursors are removed and combustion of the material mainly containing Precursor B gives rather small amounts of chlorinated organics.

Another report [11] relates the amount of HCl present in the incinerator stack fumes to the concentration of PCDDs and PCDFs found in the emissions. It would seem that the higher the concentration of HCl present in the fumes the higher the concentration of chlorinated organics. Table 21 shows exactly this relationship.

Table 21
Cencentrations of PCDDs, PCDFs and HCl in
Emissions from Municipal Incinerators
Burning Various Type Materials

Incinerators	He-CDD -CDF (ppb)	Hp-CDF (ppb)	Hp-CDD (ppb)	OCDF (ppb)	OCDD (ppb)	HCl (ppm)
A_1	90	68	145	65	152	10-200
A_2	39	30	42	31	118	10-200
A_3	135	50	55	35	90	10-200
B	—	—	6	—	12	0-10
C	—	—	5	—	5	0-10

A = inc. withot any treatment
B = inc. after recycling
C = inc. after compost production.

In December, 1980, A. D. Little, Inc. issued a report for the American Society of Mechanical Engineers on the "State-of-the-Art of Dioxin from Combustion Sources" [66]. This was done because of the great concern by the solid waste resource recovery industry regarding the presence of 2,3,7,8-TCDD in particulates. This report does a respectable job of summarizing the published literature on the status of knowledge regarding all facets related to PCDD emissions - identifying all documented combustion sources; the chemistry of PCDDs; the sampling, extraction,clean-up of extracts, and analysis for PCDDs; and toxicological considerations; as well as conclusions and recommendations. Naturally, this was most helpful in assuring that we covered the literature thoroughly up to June, 1980.

In 1981, Redford of the EPA and co-workers [95] collected gaseous, aqueous, and solid samples of the emissions from a municipal incinerator burning 100% raw refuse and one burning 85% coal and 15% refuse. Table 22 gives the results of analyses of the purified extracts of the flue gas by capillary HRGC/HRMS in the SIM mode. No PCDDs or PCDFs were found in the extracts of fly or bottom ash or aqueous emissions. Also no PCDDs or PCDFs were found in any of the samples from the coal/refuse derived fuel (RDF) facility.

protocol requiring much handling and analyses by two different laboratories, which obviously did not agree. This suggests that the extraction procedure or analytical methods leave something to be desired. The authors hint at a possible inefficient extraction procedure.

Also in 1982, Gizzi, et al, [32] analysed the stack emissions from a modern municipal incinerator in Como, Italy. Samples were taken over a nine month period on two consecutive days each month. Sampling method, extractions, and clean-up of the extracts were state-of-the-art. Analyses were performed by packed column GC/MS in the SIM mode, the data being comparative but not strictly quantitative. Tables 25 and 26 show the concentrations of PCDDs and PCDFs in the emissions.

Table 25
Concentrations of PCDDs in Emissions
from a Municipal Incinerator in Como, Italy (ng/Nm^3)

Sample	Tetra	Penta	Hexa	Hepta	Octa	Total
1	17 (14.9)[a]	n.d.[b]	18 (15.8)	15 (13.2)	64 (56.1)	114
2	< 32	n.d.	97 (37.5)	65 (25.0)	97 (37.5)	259
3	19	30 (14.0)	89 (41.6)	45 (21.0)	50 (23.4)	214
4	683 (16.3)	701 (16.8)	1391 (33.2)	1045 (25.0)	365 (8.7)	4185
5	1127 (10.7)	2089 (19.8)	3805 (36.1)	2884 (27.4)	631 (6.0)	10536
6	31 (11.1)	37 (13.2)	66 (23.5)	73 (26.1)	73 (26.1)	280
7	12 (7.9)	n.d.	42 (27.6)	37 (24.4)	61 (40.1)	152
8	10 (6.7)	n.d.	26 (17.4)	43 (28.9)	70 (47.0)	149
9	134 (15.9)	230 (27.2)	290 (34.4)	125 (14.8)	65 (7.7)	844
10	23 (12.4)	52 (27.9)	69 (37.1)	19 (10.2)	23 (12.4)	186
11	43 (4.1)	112 (10.8)	161 (15.5)	351 (33.8)	371 (35.8)	1038
12	7 (9.8)	13 (18.3)	19 (26.8)	13 (18.3)	19 (26.8)	71
13	12 (13.1)	20 (21.7)	24 (26.1)	14 (15.2)	22 (23.9)	92
14	15 (12.2)	23 (18.7)	31 (25.2)	21 (17.1)	33 (26.8)	123
15	11 (13.1)	19 (22.6)	15 (17.9)	16 (19.0)	23 (27.4)	84
16	19 (7.0)	35 (12.9)	46 (16.9)	63 (23.1)	109 (40.1)	272
17	13 (7.6)	23 (13.5)	33 (19.3)	38 (22.2)	64 (37.4)	171
Mean[c]	128.4	b	366	286.3	125.9	1104.1

[a] The numbers in brackets give the percentage of the individual classes of isomers.
[b] n.d. - Sensitivity limits not determinated because no analytical standard was available. For the same reason average values for P_5CDDs have not been calculated.
[c] The mean was calculated assigning a value equivalent to one half the detection limit to samples 2 and 3.

Table 26
Concentrations of PCDFs in Emissions
from a Municipal Incinerator in Como, Italy (ng/Nm^3)

Sample	Tetra	Penta	Hexa	Hepta	Octa	Total
1	-[a]	-	-	-	-	-
2	94 (11.1)[b]	130 (15.4)	204 (24.2)	245 (29.0)	171 (20.3)	844
3	152 (17.7)	171 (20.0)	198 (23.1)	276 (32.2)	60 (7.0)	857
4	1046 (26.2)	620 (15.5)	890 (22.3)	692 (17.3)	749 (18.7)	3997
5	2846 (28.9)	2261 (23.0)	2928 (29.8)	1414 (14.4)	382 (3.9)	9831
6	56 (10.0)	74 (13.3)	177 (31.7)	141 (25.3)	110 (19.7)	558
7	25 (7.8)	54 (16.9)	73 (22.8)	75 (23.4)	93 (29.1)	320
8	17 (10.6)	17 (10.6)	25 (15.6)	47 (29.4)	54 (33.8)	160
9	288 (31.6)	214 (23.5)	164 (18.0)	178 (19.5)	68 (7.4)	912
10	53 (25.5)	46 (22.1)	39 (18.8)	36 (17.3)	34 (16.3)	208
11	102 (21.3)	103 (21.5)	101 (21.0)	100 (20.8)	74 (15.4)	480
12	30 (23.6)	27 (21.3)	22 (17.3)	29 (22.8)	19 (15.0)	127
13	24 (21.1)	25 (21.9)	23 (20.2)	25 (21.9)	17 (14.9)	114
14	55 (24.0)	57 (24.9)	37 (16.2)	41 (17.9)	39 (17.0)	229
15	36 (21.7)	52 (31.3)	25 (15.1)	31 (18.7)	22 (13.2)	166
16	74 (20.9)	93 (26.3)	80 (22.6)	64 (18.1)	43 (12.1)	354
17	46 (19.2)	60 (25.0)	41 (17.1)	48 (20.0)	45 (18.7)	240
Mean	309	250.3	314.2	215.1	123.8	1212.3

[a] Sample 1 was not analyzed because PCDF standards were not available at that time.
[b] The numbers in brackets give the percentage of the individual classes of isomers.

It is worth emphasizing that considering the stack flow (~ 33,000 $Nm^3/hr.$) on the days of samplings 4 and 5, about 2g each of PCDDs and PCDFs were released during 8 hours each day. On these days, the incinerator operating temperature was the lowest (~ 500°C.), probably because of the high moisture content of the waste, which contained a lot of vegetables, and because of rainy weather.

In 1983, examination of the emissions from municipal incinerators continued strongly, but not much significantly new knowledge was gained.

Chiu, et al, [17] analysed emissions from two Canadian municipal incinerators (a large, modern heat recovery incinerator and a small, modular controlled-air one) along with coal-fired power and heating plants. The sample preparations and clean-up procedures are well established and analysis was performed by HRGC/LRMS using negative oxygen chemical ionization in the MID mode. Table 27 gives the analytical results, which contain no surprises.

B. Industrial Waste Incinerators

The first reported findings of PCDDs in an industrial waste incinerator was by Buser and co-workers in 1978 [80,81]. They analysed the fly ash from an industrial heating facility burning used industrial oils near Aarau, Switzerland. The sampling of the fly ash, its extraction and clean-up of the extract were state-of-the-art. Analysis was by capillary HRGC/MS with EI source and SIM technique-certainly state-of-the-art. Table 36 gives the analytical results on the fly ash. This is one of the few reports that shows DCDDs and TrCDDs formed by combustion. Chlorobenzenes and chlorophenols were also identified in the fly ash. The amounts of PCDDs and PCDFs were higher in the fly ash from this industrial waste incinerator than in a municipal incinerator.

Table 36
PCDDs and PCDFs in Fly Ash from
a Switzerland Industrial Waste Oil Incinerator (ppb)

DCDD	10	TCDF	100
TrCDD	20	PeCDF	100
TCDD	100	HCDF	70
PeCDD	160	HpCDF	50
HCDD	180	OCDD	n.d.
HpCDD	130		
OCDD	40		

n.d. = not detectable

The Dow reports of 1978, 1980 and 1982 on the "Trace Chemistries of Fire" [13,23] previously mentioned confirm the findings of Buser and co-workers. Dow analysed the particulates from a stationary tar burner and a rotary kiln incinerator at their Midland, Michigan plant. Their analytical methodology was state-of-the-art as previously discussed. Table 37 gives the detailed results of their analysis for PCDDs [66] [see Table 12 (p.14) for a comparison of analytical results with other combustion sources]. A very dramatic feature of the data in Table 37 is the reduction in total PCDD levels when clean supplementary fuel, rather than tarry waste, is burned in the secondary combustion chamber of the rotary kiln incinerator. The supplemenatary fuel causes a higher temperature in the secondary combustion chamber, thus PCDDs are more completely destroyed or cannot form to as great an extent.

In 1981, Hryhorczuk and co-workers [38] examined scrapings from two of three stacks and from all three furnaces of a wire reclamation incinerator in the U.S. midwest. The samples were analysed for TCDDs and TCDFs by Gross of the University of Nebraska using state-of-the-art GC/HRMS techniques. Table 38 gives the results on two of the ash samples. The authors indicated that these compounds could have entered the incinerator intact in the fuel or charge, or could have formed from precursors in the wire insulation.

Table 38
TCDDs and TCDFs in Ash from
a Wire Reclamation Incinerator (ppt)

Sample	Total TCDDs	Total TCDFs
Furnace No. 2	58	730
Stack No. 2	410	11,600

In 1982, Barnes, et al,[7] reported that the U.S. EPA, in 1979, carefully examined the emissions of two commercial incinerators used to burn PCB wastes. In each case, detectable levels of PCDDs and PCDFs were found.

Also in 1982, Rappe, et al,[94] analysed a series of samples taken during a test study of the thermal decomposition of PCBs in a rotary cement kiln in Norway. The temperature of the oven was at 1450°C. No PCDDs or PCDFs (tri-to octachloro isomers) could be found in any of the samples. This suggests that this might be a safe way to destroy PCB containing wastes.

In 1983, Tiernan and co-workers [101] investigated the combustion products from incineration of chlorinated chemical wastes. Table 39 shows the analytical results along with the concentration of CDDs/CDFs in the original PCP wastes incinerated.

Table 39
Relative Concentrations of CDDs/CDFs in Chemical Wastes
and Combustion Products Thereof[a]

CDD/CDF Chlorinated Class	4,5-Pentachlorophenol (PCP) in Oil		PCP Sludge from Wood Treatment Plant		Ash from Combustion of PCP Wastes		Flue Gases from Incineration of Mixtures of Chlorinated Chemical Wastes	
	CDDs	CDFs	CDDs	CDFs	CDDs	CDFs	CDDs	CDFs
Tetra-	0.00028	0.0099	--	--	0.018	0.88	0.80	0.17
Penta-	0.0083	0.076	0.0013	0.023	0.16	1.0	0.80	1.0
Hexa-	0.14	1.0	0.0055	0.062	0.41	0.63	0.20	0.83
Hepta-	0.064	0.063	0.31	1.0	0.59	0.75	0.40	0.40
Octa-	1.0	0.39	1.0	0.25	1.0	0.25	1.0	0.33

a. Normalized to chlorinated class detected in largest concentration.

These data suggest that some degradation of the higher CDDs/CDFs present in the wastes occurred during combustion with an increase in the relative concentrations of the lower CDDs/CDFs (particularly the Cl_4 and Cl_5 isomers). The distribution of CDD/CDF homologs (isomer clusters) in the mass spectrum is distinctly different from those observed in the flue gases from municipal incinerators, but the major TCDD isomers were the same (1,3,6,8-, 1,3,7,9-, and 1,3,7,8-TCDD).

Finally, at the National ACS Meeting in Washington, D. C. last September (1983), Gross, et al,[122] investigated the incineration of chemical wastes. They measured the TCDD and TCDF content of the starting chemical wastes and in the emissions on incineration. The [illegible] in Tables 40 and 41. Except for one emission

C. Oil/Coal-fired Powerhouses and Utility Facilities

The first report of PCDDs detected in oil/coal-fired powerhouses or utility facilities was by Dow in 1978 [66] in their original "Trace Chemistries of Fire" paper. The study was elaborated on in publications of 1980 [13] and 1982 [23]. Dow analysed their Midland, Michigan powerhouse operating under normal conditions with fuel oil and coal. The results are reported in Table 12 (p.14). With regard to HCDDs through OCDD, the concentration of these PCDDs in the emissions from the powerhouse is the lowest of the incinerator type sources; however, the concentration of TCDDs appears to be the highest compared to the others. This seems unusual.

In 1980, Kimble and Gross [39] analysed fly ash from the stack of a 750 MW power plant burning low sulfur, high ash (10%) coal with 50 ppm chlorine content. Quantitative analysis using packed column LRGC/HRMS with SIM mode showed no detectable TCDDs at a detection limit of 0.6 ppt. They did not analyse the fly ash for other CDD homologs. This detection limit is lower by a factor of at least 30,000 than that determined by Dow in their study. Kimble and Gross say that one explanation for the large difference in TCDD concentration may be the nature of the fuel sources. For this reason, the authors cautioned that the finding of significant amounts of PCDDs in emissions from municipal and chemical waste incinerators should not be used to infer that these compounds are also significant products of fossil-fueled power plants. Thus their results contradict Dow's findings.

Also in 1980, Lustenhouwer, Olie and Hutzinger [84] showed that the extraction procedure used by Kimble and Gross gives poor recoveries for PCDD. They also analysed fly ash from a coal-fired powerhouse with methodology suitable for the detection of ppb levels of chlorinated compounds. PCDDs were not detected but they did find chlorobenzenes in the fly ash. The authors did not comment whether oxygen was in excess or not.

In 1981, Junk and Richard [46] analysed particulates and gaseous emissions from a utility facility producing 35 MW of electrical power from steam produced by co-burning coal and refuse derived fuel (RDF). Using HRGC/MS with MID technique with detection limits of 0.1 ppb for particulate samples and 0.5 ppt for vapor, no TCDDs were detected in the emissions. The influent coal and RDF also showed no detectable TCDDs. They explain no detectable TCDDs in the emissions by (1) the high operating temperature of 1200°C. due to the coal supplement and the type of boiler (2) sufficiently long resident time (>1.3 sec.) (3) adequate O_2 (4) small pieces of RDF produced by shredding. These four conditions have not existed for most incineration processes where TCDDs have been found. They say the one exception may be the negative TCDD results by Kimble and Gross with a presumed higher operating temperature. Prior to this study, very little data on the operating conditions of the various municipal incinerators and power plants where the emissions were found to contain PCDDs had been published.

In 1981, Redford, et al, [95] examined the gaseous, aqueous, and solid emissions of a utility facility burning 85% coal and 15% RDF. No PCDDs or PCDFs were detected using capillary GC/HRMS with SIM technique at a detection limit of 10 ppt. This finding is thus in agreement with those of Junk and Richard, Kimble and Gross, and Lustenhouwer.

In 1982, Krishnan and Hellwig [55] surveyed the literature regarding trace emissions from various oil/coal-fired type boilers. Using a set of assumptions, national emissions of the trace pollutants were estimated. A "dioxins" emission factor was only reported for coal-fired utility boilers (0.01 ppt/Joule of energy input). This translated to an estimated U.S. national emission in 1978 of ~220 lbs. of "dioxins"/yr. This is considerably less than all other estimated trace emissions, such as metals or organics which are known or suspected carcinogens [As, Be, Cd, Hg, formaldehyde and benzo(a)pyrene]. This survey indicates that coal combustion emits significant quantities of a greater number of trace pollutants than does oil combustion.

In 1983, Ahlberg,et al,[1] analysed the particulates from an oil-fired boiler fired with heavy fuel oil (ash, 0.06%; sulfur, 2%) and a pulverized coal-fired boiler fired with Polish coal (ash, 0.13%; sulfur, 0.8%) for 2,3,7,8-TCDD and TCDF. In both cases, neither compound was detected with detection limits at 75-140 pg/g dry fuel for 2,3,7,8-TCDD and 12-22 pg/g dry fuel for 2,3,7,8-TCDF. State-of-the-art procedures for sampling, extraction and cleanup of the extractants were used. Analysis was by GC/HPMS with SIM technique. More organics were found in the oil soot than in the coal fly-ash.

Also, in 1983, Chiu, et al, analysed fly ash from a large, modern coal-fired thermal generating station and from a 30 year old coal-fired boiler at a central heating plant. Sampling, extraction, clean-up and analysis were state-of-the-art. Analysis was performed by HRGC/LRMS using negative oxygen chemical ionization source with MID technique. Table 42 gives the analytical results.

Table 42
PCDDs and PCDFs in Coal-fired Power Plants (ppb)

	Cl_4		Cl_5		Cl_6		Cl_7		Cl_8	
Fly ash Source	CDD	CDF	CDD	CDF	CDD	CDF	CDD	CDF	CDD	CDF
Modern power plant	1	8	6	32	6	18	5	18	-	-
30 yr.old central heating plant	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd

nd = not detected

Regarding the modern power plant, the authors indicated there was not enough data to conclude whether it does or does not represent a potential source of PCDDs/PCDFs. The data reported above is from only one fly ash sample. Other samples showed only non-measureable traces of these compounds.

Also in 1983, Eklund and Stromberg [28] analysed the emissions from a coal-fired fluidized bed combustor and a coal-fired automatically regulated, on/off boiler for greenhouse heating as well as a fluidized bed municipal incinerator. The authors indicate that PCDDs and PCDFs were found in flue gas samples from the municipal incinerator but makes no mention of their presence or absence in the emissions from the coal-fired power units. Analysis was performed by GC/MS but no quantitative data are given. The data and the article, per se, are judged poor.

Finally, as mentioned earlier, Haile and co-workers[33] analysed the emissions from the Ames, Iowa Municipal Coal Boiler Power Plant. Many compounds which could be precursors to PCDDs and PCDFs, such as PCBzs, PoCPs, and PCBs, were quantified but no PCDDs or PCDFs were found at a detection limit of 0.1-0.25 ng/dscm.

Related to the above studies, Mahle and Whiting [67] reported some laboratory studies dealing with the pyrolysis of bituminous coal at 600°C. in the presence of NaCl, HCl and Cl_2. Table 43 gives the results. This investigation clearly shows that combustion of coal under certain conditions can yield significant quantities of PCDDs.

Regardless of Mahle and Whiting's findings, it appears that, in general, oil- and coal-fired power plants and utility facilities are unlikely sources of significant PCDD and PCDF emissions.

D. Wood Burning Fireplace/Furnaces

Here again, the first report of PCDDs detected in wood burning fireplaces and furnaces was by Dow in 1978 [66] in their original "Trace Chemistries of Fire" paper. Elaborations of this study were published in 1980 [13] and 1982 [23]. Dow analysed the carbonaceous material on the walls of a metal firebox of a 25 year old fireplace, whose flue had not been cleaned in 10 years. Burch, oak, willow, mountain ash and applewood untreated with preservatives (such as pentachlorophenol) had been burned along with some paper to start fires. Also the residue on firebrick in a 12 year old fireplace, whose chimney had never been cleaned, was sampled. Hardwood, mostly oak, untreated with preservatives, was used as fuel. The results are shown in Table 12 (p.14). The levels of TCDDs and HCDDs are quite small while HpCDDs and OCDD are present in more significant quantities. The A. D. Little report [66] suggests that the findings in the wood burning fireplaces and other commonplace samples seem tenuous when judged by the

standards of the rest of the Dow report. The history of the sampled fireplace walls and mufflers is not well documented. The reported observations may reflect circumstantial, rather than causal, relations between the apparent PCDDs and the commonplace combustion process. Also, for the auto mufflers and the charcoal grilled steaks, the identity of the PCDD species reported was not confirmed by GC/MS to be present at levels exceeding two times the detection limit.

In 1982 [71] and 1983 [70], Nestrick and co-workers reported on a study to determine if PCDDs were produced during the combustion of wood. They reasoned that if PCDDs can be detected from its combustion today, PCDDs have been present in the environment from at least as far back as the appearance of trees. Residential wood combustion (RWC) was selected as the source of particulate emissions, while uncontrolled open-burning situations (i.e., forest fires) or open-burning experiments were ruled out for various reasons. All combustion particulate samples were obtained from domestic heating systems. Preliminary evaluation samples were obtained from the flues of an exclusive oil heating system and an exclusive wood heating system located in Cape Cod, MA as well as a primary wood heating system located ~ 15 miles from Midland, Michigan. For the actual study, six individual samples of chimney particulates were collected from each of three rural regions in the U.S. No particulate samples were obtained from systems where any type of treated or manufactured wood was burned or where the wood or residence had ever been exposed to materials like 2,4-D, 2,4,5-T or pentachlorophenol. The Eastern regional samples were taken from residences near Farmington, NH, an agricultural community. The Central regional samples came from near Grand Marais, MN (a tourist and logging area), at an old farm site near Duluth, MN, and near Escanaba, MI in the upper peninsula. The Western regional samples came from a rural area near Creswell, OR. State-of-the-art sampling, extraction, clean-up and analytical methodology were employed. Reverse phase HPLC was employed in the clean-up procedure to assure removal of residual chemically similar interferences to PCDDs. Both HRGC/LRMS and HRGC/HRMS in the SIM and MPM (multiple peak monitoring) mode with several standards were used for isomer specific PCDD analysis. Table 44 gives the results of the preliminary evaluation sampling.

Table 44
PCDDs in Combustion Particulates from Preliminary Experiments(ppt)

	flue pipe particulates,[a] Cape Cod, MA		RWC unit particulates, Midland, MI[a]			
	exclusive oil burner	exclusive wood burner	chimney[c] base	flue pipe back	flue pipe front	firebox ash
total TCDDs	ND[b] (2.4)	260	950	7.5	2.9	1.6
total HCDDs	ND (4.2)	340	3400	17	16	ND (2.0)
total H_7CDDs	25	420	8100	89	81	30
OCDD	130 (20)	260	12000	250	242	120
total CDDs	155	1280	24450	363.5	341.9	151.6

[a] Refer to text for sample descriptions. [b] Not detected, refer to footnote *d* in Table46 [c] Isomer-specific results presented in Table 45

The following comments and conclusions were given:

(1) For all regional samples in which one or more TCDD isomer was observed, the % 2,3,7,8-TCDD relative to total TCDD was 0-16% with an average of 5%. This average composition for 2,3,7,8-TCDD is representative of rural regions and is similar to the 3.3% 2,3,7,8-TCDD observed for the Midland RWC chimney particulates where locally obtained wood was from an industrialized area associated with production of chlorinated compounds.

(2) PCDD formation appears to be related to the natural chlorine content of the wood fuel rather than environmental sources. In a published study by TRW [133], eleven different varieties of wood were quoted to contain chlorine concentrations of 14-84 ug/g.

(3) Typical residential wood combustion units located in rural areas are a source of PCDD emissions to the environment. The observation of a relatively flat distribution pattern for TCDD isomer concentration suggests that their formation in wood fueled RWC units may be a random statistical process where each of the 22 TCDD isomers is equally favored.

(4) Although these results suggest that wood combustion may be a potential significant contributor to environmental PCDD background levels, additional research is necessary to confirm "The trace chemistries of fire" hypothesis and to better characterize the magnitude of this source.

(5) Because it has been estimated that particulate emissions from forest fires in the U.S. alone range from 0.5×10^6 and 54×10^6 tons/year, the authors find it difficult to believe that PCDDs are not ubiquitous in the environment.

Also PCDFs were found to be major components of these flue particulates. The apparent isomer distribution for TCDFs is similar to that of TCDDs, in that a large number of isomers are present at reasonably similar levels. The fact that unchlorinated dibenzofuran was identified whereas unchlorinated dibenzo-p-dioxin was not, may indicate that PCDDs and PCDFs are produced by different mechanisms during wood combustion. It also may indicate that the unchlorinated dibenzofuran ring system is more thermally stable than unchlorinated dibenzo-p-dioxin. PCDD concentrations increase with increasing degree of chlorination whereas PCDF concentrations increase with decreasing degree of chlorination.

Also in 1982, Smith,et al,[98] analysed a sample of common wood-burning fireplace soot from Clifton Park, New York for 2,3,7,8-TCDD and TCDF. Extraction and clean-up procedures were state-of-the-art. Analysis was performed using RP-HPLC and capillary column GC/HRMS with ion monitoring technique. The analytical results indicated 2.7 ppb 2,3,7,8-TCDD and 7.2 ppb 2,3,7,8-TCDF. The results seem rather tenuous compared to the other data given in the report.

E. Diesel/Auto Exhausts

This potential source has had very limited attention, but could have very significant repercussions. The Dow articles [13, 23] suggest that the quantities of PCDDs emitted are very small [see Table 12, (p.14)] Mufflers from gasoline-powered automobiles were collected at muffler shops in Pontiac and Detroit, Michigan (more than 100 miles from Midland), as they were removed from cars. The car owners voluntarily gave the mufflers and information about mileage and gasoline type used. The make, model, and mileage of each car were noted. The mufflers were transported to the Dow Analytical Laboratories sealed in plastic bags. Mufflers from Diesel-powered trucks were collected at Auburn and Saginaw, Michigan. They were sealed and transported as above. At the Dow Analytical Laboratories, the inside chambers of the mufflers were scraped and residue was collected from these chambers and analyzed for chlorinated dioxins by both GC with electron capture detection and GC/MS after liquid chromatographic separation.

The only other article dealing with PCDDs in Diesel/auto exhaust emissions was published by Cavallaro, et al, [82] in 1980. They mention that only OCDD was found in an auto exhaust sample. No quantitative data was given. The extraction, clean-up and analytical procedures appear to be state-of-the-art. Analysis was performed using capillary HRGC/MS with a MNCI (methane negative ion chemical ionization) source.

With these "hints" of possible PCDD emissions from truck/auto exhausts, one wonders why more publications have not appeared on this subject. These two articles suggest that the quantities of PCDDs emitted are very small (if at all) and, it could be, that researchers in this field feel that this is not a significant emission source. Because of its potential significance, more studies in this area are warranted.

F. Miscellaneous Sources

The search of the literature revealed several other possible non-industrial sources of PCDDs, all of them being combustion processes. These include cigarette smoke, charcoal grill fumes, and accidential fires, particularly those involving PCBs.

The only references to the possible presence of PCDDs in cigarette smoke and charcoal grill fumes are the Dow reports [13, 23]. The analytical results are shown in Table 12 (p.14).

In cigarette smoke, trace quantities of hexa-, hepta- and octa-CDDs near the detection limits were found. Smoke generated by the simulated smoking of cigarettes (an arbitrarily selected national brand without filters) was analyzed for trace levels of chlorinated dioxins. The smoke was collected on special collection matrices consisting of 100/120-mesh silica (coated in situ with 20 percent hexadecane by weight) contained in a disposable glass transfer pipette. The cigarette was attached to the pipette by cleaned latex rubber tubing and puffed by applying gentle vacuum with a rubber pipette bulb.

Approximately 20 to 30 puffs nominally 2 to 3 seconds in duration were collected from each cigarette. An entire package of 20 cigarettes was combusted during each experiment. Combustion experiments were conducted in two different urban locations. Before use, the inlet end of each collection tube was spiked with 2.0 nanograms of ^{13}C-labeled 2,3,7,8-TCDD, which served as an internal standard for TCDD quantitation after residue cleanup. Each collection tube was extracted with hexane, followed by a multistep cleanup, including reverse phase LC, to remove interferences. The LC eluate was subjected to both low resolution GC/MS and electron capture GC analysis.

Concerning charcoal grill fumes, only trace quantities of OCDD were found near the detection limit. New York strip steaks grilled over a charcoal fire were sampled at 13, 18, and 25 minutes (rare, well-done and overdone samples). Each of the three samples was immediately wrapped in aluminum foil, placed in a plastic bag, and put in a freezer to await analysis. Sample extraction and clean-up procedures were state-of-the-art. Analyses of the extracts were performed by high pressure liquid chromatography and low resolution GC/MS. The data is certainly tenuous.

Published reports on PCDDs and PCDFs found in the fumes from accidental fires are more numerous. The first reference to PCDDs found in an accidental fire was a report by Harvan,et al, [34] in 1981, who collected air filter samples from an industrial fire in Elizabeth, NJ. The samples were extracted and the extracts cleaned up in an acceptable manner. Analysis was by HRGC/HRMS with MID and CID techniques. Analytical complications arose due to chemical interferences. The researchers concluded that, of the nine samples taken, no sample contained more than 40 pg of TCDDs and that one sample contained ~ 20 pg of a specific TCDD isomer. Four of the samples contained less than 5 pg of TCDDs.

From 1982 to the present, several reports have been published on an electrical fire that occurred in the State Office Building in Binghamton, NY. A transformer, which contained about 1100 gallons of Pyronox (a 55% mixture of PCBs and 35% PCBzs) was damaged by the fire. About 180 gallons were lost. How much was burned and how much leaked before it was cleaned up is not known. As a result of the fire, a large amount of soot was formed. Kim [51] reported the level of 2,3,7,8-TCDD in the soot was ~ 3ppm. This data appears to come from a more scientific article published by Smith, et al. [98] A composite sample of soot from a stairwell of the building was analysed. The extraction and clean-up procedures were state-of-the-art. Analysis was by RP-HPLC followed by capillary column GC/HRMS with ion monitoring techniques. Analysis of duplicate samples gave 2.8 and 2.9 ppm of 2,3,7,8-TCDD, and 270 and 120 ppm of 2,3,7,8-TCDF. des Rosiers, [42] of the EPA Office of Res. & Dev., reported, however, that the analysis of soot samples found in the building shows PCDDs as high as 10-20 ppm while PCDFs were present as high as 2,160 ppm. He said the most toxic PCDDs and PCDFs were the major components in the soot, including 2,3,7,8-TCDD, 2,3,7,8-TCDF, 1,2,3,7,8-PeCDD, and 1,2,3,7,8- and 2,3,4,7,8-PeCDFs. des Rosiers also said that even after parts of the building were decontaminated using steam and detergents, trace amounts (ng/m^2) of PCDDs and PCDFs were still present.

The data in the report by des Rosiers seem to have come from some outstanding work by Rappe and co-workers. [94,123] In addition to analysing soot from the Binghamton, NY fire, these researchers analysed samples from PCB fires at Stockholm, Skövde, and Surahammar, Sweden as well as an explosion of a capacitor filled with PCB in an electrical railway locomotive. The toluene Soxhlet extraction of the acid treated samples and the clean-up of the extracts were state-of-the-art. The analyses were performed by HRGC/MS using EI or MNCI sources and SIM technique with reference standards.

In the Binghamton, NY soot, the total level of PCDFs was 2,160 ug/g(ppm). The most toxic isomers, as mentioned above, were found to be the major constituents. In addition, a series of PCDDs were found with the highly toxic isomers again dominating (2,3,7,8-TCDD at 0.6 ppm and 1,2,3,7,8-PeCDD at 2.5ppm). Most interesting was the finding of polychlorinated biphenylenes (PCBPs). The PCBPs are closely related to the PCDFs and PCDDs and the 2,3,6,7-TCBP has biological activity in the same range as 2,3,7,8-TCDD [124].

In the Stockholm capacitor fire, the wipe samples were found to contain PCDFs and PCBPs but no PCDDs. In the Skövde capacitor fire, the capacitor fluid was a mix of mineral oil and PCBs. The wipe samples contained PCDFs but no PCDDs or PCBPs. The Surahammar explosive fire and the electric locomotive fire also generated only PCDFs.

Table 50 gives the analytical results.

Table 50
PCDFs, PCDDs and PCBPs in Soot from
PCB Capacitor Fires

	PCDFs							
	ΣCl_4	2378	ΣCl_5	ΣCl_6	ΣCl_7	Cl_8	Σ PCDDs	PCBPs
Binghamton (μg/g)	20	12	670	965	460	40	20	+
Stockholm (μg/m²)	1.2	0.15	0.175	-	-	-	-	+++
Skövde (μg/m²)	0.6	0.1	0.1	0.06	0.008	0.006	-	-
Surahammar (μg/m²) (before cleaning)	1.2	0.3	0.3	0.15	0.07	0.01	-	-
Surahammar (μg/m²) (after cleaning)	< 0.02	< 0.004	< 0.01	< 0.01	< 0.015	< 0.002	-	-
Hallstahammar (μg/m²) (before cleaning)	1.6	0.54	0.36	0.60	0.80	1.34	-	-
Hallstahammar (μg/m²) (after cleaning)	0.002	0.0002	0.0005	0.0003	0.0007	0.0011	-	-
Electrical locomotive (μg/m²)	4.9	1.2	1.5	0.4	0.17	0.04	-	-

Also, Nestrick, et al, [70,71] in their excellent research on emissions from residential wood combustion (RWC) units admitted that none of the combustion sources in Dow's "Trace Chemistries of Fire" paper truly demonstrates situations representative of "common combustion" in a time scale which includes the "advent of fire". These researchers reasoned that if PCDDs can be detected from wood combustion today, PCDDs have been present in the environment from at least as far back as the appearance of trees. With the finding of PCDDs in RWC emissions, they concluded, "Because it has been estimated that particulate emissions from forest fires in the U.S. alone range between 0.5×10^6 and 54×10^6 tons/year, we find it difficult to believe that CDDs are not ubiquitous in the environment."

Finally, Czuczwa and Hites have recently [119,120] reported on a classic study of PCDDs and PCDFs in sediment cores. The results are most significant. PCDDs and PCDFs were found in sediment samples in an urban area as well as in remote sites, the concentration at the remote sites being about 100 ppt. This indicates that PCDDs and PCDFs are stable in the environment and "indeed, may be ubiquitous". The PCDD and PCDF isomer profiles were similar in all sediment samples and closely followed those found in combustion samples. This suggests that combustion is the major source of the compounds. When PCDDs and PCDFs were measured in sediment cores, the depth-concentration profiles in these cores showed that the input of PCDDs and PCDFs increased dramatically at a depth corresponding to about 1940 and remained high to the present time. In Figure 2, the time-dependent concentration profiles of OCDD and HpCDF in a Lake Huron core are compared to the trends for U.S. coal production and production of chlorinated hydrocarbons for that time period. It seems clear that coal use has not been a major source of these materials at least since 1940. The sedimentary historical record agrees well with the production of chlorinated aromatic compounds. According to the authors, this suggests that chlorinated aromatics are precursors of PCDDs and PCDFs, and these compounds are present in combustion fuels. The following statement made by the authors when presenting this information at the 186th National ACS Meeting in Washington, D. C. in September, 1983 caused much argument: "Low or undetectable levels of PCDDs and PCDFs at depths corresponding to times before 1940 show that PCDDs and PCDFs were not with us since the advent of fire." This statement was challenged for several reasons:

1. The authors did not demonstrate that PCDDs and PCDFs on the particles had always been there.

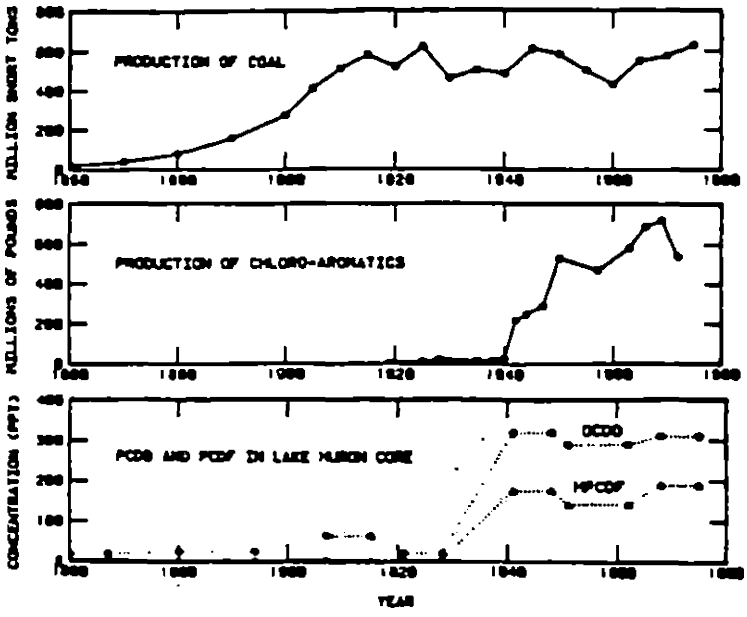
2. The sample population was very small.

3. Precision values were not available.

4. The data given showed a background level of PCDDs negating the claim.

5. The rise of the chemical industry parallels the rise in use of municipal incinerators, industrial waste incinerators, fireplaces, etc.

Figure 2
U.S. Coal and Chloro-aromatics Production Compared to the Concentration of PCDDs and PCDFs in Lake Huron Core Sample as a Function of Time



After considering all of the information and evidence, we believe that PCDDs as well as PCDFs are indeed ubiquitous now and have been so for a long time.

> Trace chemistries of fire consist of numerous chemical reactions occurring during combustion at very low concentrations. Recent advances in analytical methodology now permit the products of these reactions to be measured at concentrations as low as 0.0000000001 percent (10^{-11}%) in favorable cases. With this sensitivity, products resulting from reactions with yields as low as 10^{-10} percent can be identified and measured. The chemistry is such that low concentrations of either inorganic or organic chlorides in the fuel can be expected to produce traces of chlorinated dibenzo-p-dioxins.

Table 12 (p. 14)[13] gives the data which Crummett and co-workers claim is evidence in support of their hypothesis and the ubiquitous nature of PCDDs. Eminent scientists in the U.S. have suggested other ways to test the hypothesis. Among the suggestions were:

1. Determine if chlorinated hydrocarbons and chlorinated dioxins are present in soil high in carbonaceous matter taken from drill cores at depths corresponding to 5-, 12-, and 35,000 years from under ancient lake beds.

2. Determine if chlorinated hydrocarbons and chlorinated dioxins are present in ice taken from the center of an ancient glacier.

3. Determine if chlorinated hydrocarbons and chlorinated dioxins are present in ash taken from the mouth of a volcano.

4. Determine if chlorinated hydrocarbons and chlorinated dioxins are present in sea breezes from islands remotely located in the South Pacific.

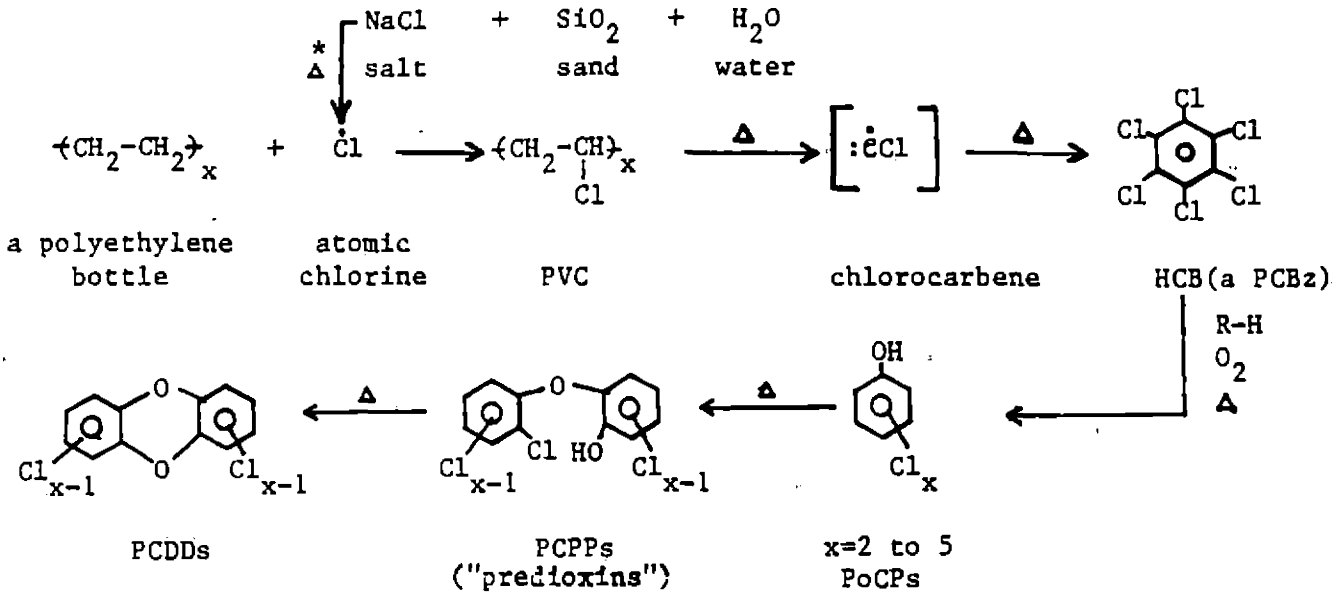
5. Determine if chlorinated dioxins can be formed by burning fossil fuel in the presence of chlorine or inorganic chloride.

6. Determine if chlorinated dioxins are present in fish taken from rivers remote from pesticide manufacturing facilities but close to incinerators and fossil-fueled power houses.

Crummett indicated that suggestions 1 through 4 would require detection limits of 1 ppq and possible contamination would make the analysis very difficult. However, Czuczwa and Hites [119,120] reported finding PCDDs and PCDFs in sediment core samples, particularly from remote sites, as far back as 1868. They said that "these data indicate that PCDDs and PCDFs are stable in the environment and, indeed, may be ubiquitous". Also the isomer profiles were similar in all core samples and closely followed those in combustion samples, suggesting that combustion is the major source of these materials.

Concerning suggestion 6, Crummett reported on the analysis of fish for TCDDs. The results are shown in Table 51. [23]

chloride may form PCBzs which, in turn, form PoCPs which proceed on to form PCDDs. Such transformations can be expressed in simple chemical form by the following reaction path:



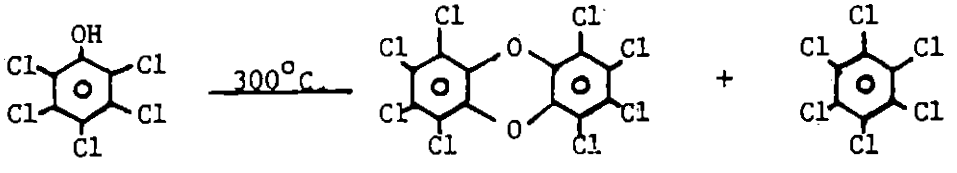
* = high heat (550-1050°C.)

Much more about the chemistry of formation of PCDDs and precursors is coming up in the next section.

B. Formation of CDDs

The production of CDDs from the pyrolysis of known chlorinated chemical compounds has been known for some time. By reviewing the findings from such experiments, one can obtain a rather clear picture of the various chemical transformations that can occur in all combustion processes.

Probably the first known production of a PCDD was in 1957 by Sandermann, et al, [126] who heated pentachlorophenol (PCP) in a tube. OCDD and hexachlorobenzene (HCB) were formed.



With the finding by Olie and co-workers [73] in 1977 of PCDDs as well as PCBzs and PoCPs in the emissions from a municipal incinerator, many researchers have performed laboratory studies over the years to determine the chemistry of formation of PCDDs. Four excellent review articles by Esposito,et al, [31], A.D. Little, Inc. [66], Lustenhouwer, et al, [84], and Choudhry and Hutzinger [18,30,85,86,127] have summarized these findings up to 1982. The articles by Lustenhouwer, et al, and Choudhry and Hutzinger are particularly good. Lustenhouwer's article is probably the first to try to discuss the possible mechanisms for the thermal generation of PCDDs and related compounds theoretically. The review by Choudhry and Hutzinger is truly a monumental piece of work covering all mechanistic aspects of the thermal formation of PCDDs and related halogenated organic compounds. Part I [18] gives the theoretical background and thermochemical decompositions of monomeric organic compounds that may take place in the "Trace Chemistries of Fire" hypothesis. Part II [30] deals directly with the thermochemical generation of PCDDs and PCDFs from closely related precursors. Part III [127] deals with the thermodegradation of organometallics which relates to the catalytic effect that certain metals may have on the formation of PCDDs, PCDFs and their related precursors. We will not discuss this area to any degree; however, such chemistry plays a role in the "Trace Chemistries of Fire" hypothesis. Part IV [85] discusses the incorporation of inorganic chlorine into organic matter and thermochemical formation of aromatic compounds like PCDDs from aliphatic organics like chloroform, polyethylene or chlorinated ethylenes. Part V covers hypotheses for the formation of PCDDs and PCDFs in combustion processes incorporating all elements discussed in the four previous articles. It discusses the so-called "de novo" syntheses of PCDDs and PCDFs, which in essence is the same as Dow's "Trace Chemistries of Fire" hypothesis.

Theoretically, the presence of a wide variety of PCDDs and PCDFs in combustion processes can be attributed to three possibilities. It is possible that PCDDs and PCDFs:

1. are trace components of refuse and do not undergo effective thermal destruction.

2. are produced during the combustion and pyrolysis of chlorinated precursors like PCBzs, PoCPs, chlorinated 2-phenoxyphenols (PCPPs) ("predioxins"), chlorinated diphenyl ethers (PCDPEs), PCBs, and 2,4,5-T and its esters. This includes the dechlorination of higher PCDDs and PCDFs to lower chlorinated isomers.

3. are formed as a consequence of a complex array of pyrolytic processes of chemically unrelated organic compounds ("de novo" syntheses) including:

   (a) anthropogenic (man-made) chlorinated organic chemicals such as polyvinyl chloride (PVC), DDT, or chloroform.

   (b) anthropogenic non-chlorinated organic such as polyethylene or polystyrene and inorganic chlorine such as salt (NaCl) or hydrochloric acid (HCl).

   (c) autochthonous (naturally occurring) organic chemicals like tobacco, coal, lignin and petroleum coke and inorganic chlorine (NaCl or HCl).

1. Ineffective combustion of PCDDs present in refuse

Concerning the hypothesis of PCDDs being in the emissions of combustion processes due to incomplete combustion of PCDDs present in the refuse, it is now apparent that PCDDs, per se, are destroyed by incineration above 800°C. [13,23] ; however, PCDDs bound to particulate matter appear to be largely unaffected by incineration temperatures as high as 1150°C. [29] Incineration tests conducted for the U.S. Air Force on Agent Orange, which contained small quantities of 2,3,7,8-TCDD, showed no TCDD in the emissions at 300 and 2 ppb detection limit. The tests were run with firewall temperatures at 1150, 1400 and 1600°C.[13] and 30% excess air. A typical municipal incinerator, such as the Chicago Northwest incinerator, operates at a flue gas temperature of 1100°C with 50% excess air, which may be borderline for combustion of any PCDDs bound to particulates in the refuse. Industrial waste incinerators are usually operated at higher temperatures by the use of supplementary fuel (coal, oil or gas), and, if operated properly, should not release significant levels of PCDDs in its emissions. The New York State Dept. of Environmental Conservation Handbook in 1980 indicates that "incineration of 2,3,7,8-TCDD at 1000°C for two seconds is believed adequate."[66]

To date, no systematic, thorough study, to our knowledge, has been carried out to determine if PCDDs or PCDFs are minor constituents of domestic refuse. These materials could enter the refuse via herbicide formulations or treated wood. However, there are three reports which suggest that PCDDs and PCDFs are not present in ordinary refuse. Liberti and co-workers [58,62] crushed, homogenized, dried and extracted a large sample of garbage to be incinerated. Analysis of the extract showed no PCDDs or PCDFs to be present. However, upon incineration, the emissions did contain PCDDs (see Table 19, p. 21). Lustenhouwer, et al, [84] analysed a sample of compost and found

From the above study by Rappe, et al, [87] , three possible routes to PCDDs from chlorophenols in combustion processes seem to be:

1. condensation of chlorophenates directly to PCDDs which includes Smiles rearrangement

2. dechlorination of higher chlorinated PCDDs

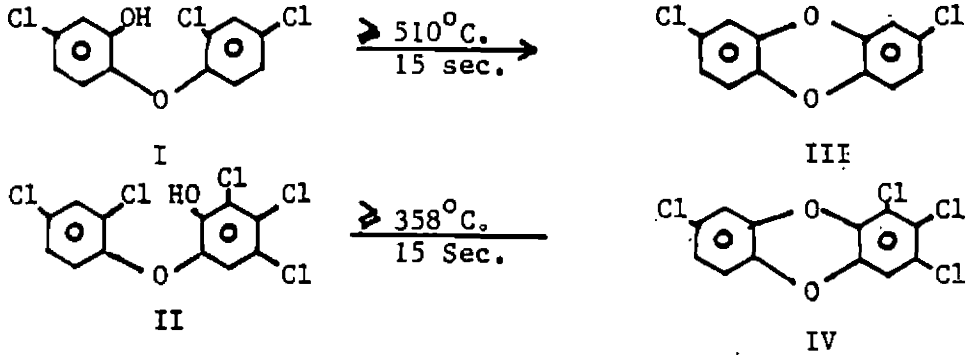
3. cyclization of chlorinated 2-phenoxyphenols (PCPPs) ("predioxins") already existing as contaminants in chlorophenates.

Route 1 is by far the most important.

c. Chlorinated 2-phenoxyphenols (PCPPs) and chlorinated diphenyl ethers (PCDPEs)

Both of these type compounds have been shown to form PCDDs upon pyrolysis. The PCPPs are also called "predioxins".

Nilsson and co-workers [128] investigated the thermal degradation of 5-chloro-2-(2,4-dichlorophenoxy) phenol (I) and 4,5,6-trichloro-2-(2,4-dichlorophenoxy) phenol (II) at various temperatures. Even at 980°C., a temperature at which most organic compounds decompose completely, MS showed only the starting materials and the products 2,8-DCDD (III) and 1,2,3,8-TCDD (IV). In a typical pyrolysis (770°C., 15 sec.), it was observed that 70%



of II degraded and 30% appeared as IV (3%) and unchanged II (27%). Table 58 gives the results of this experimentation.

Table 58
Pyrolytic Formation of PCDDs from PCPPs

Temp., °C	PCDD formed (%) * from I	II
358	0	0.4
510	1.7	0.8
770	6.0	3.0
980	6.2	2.8

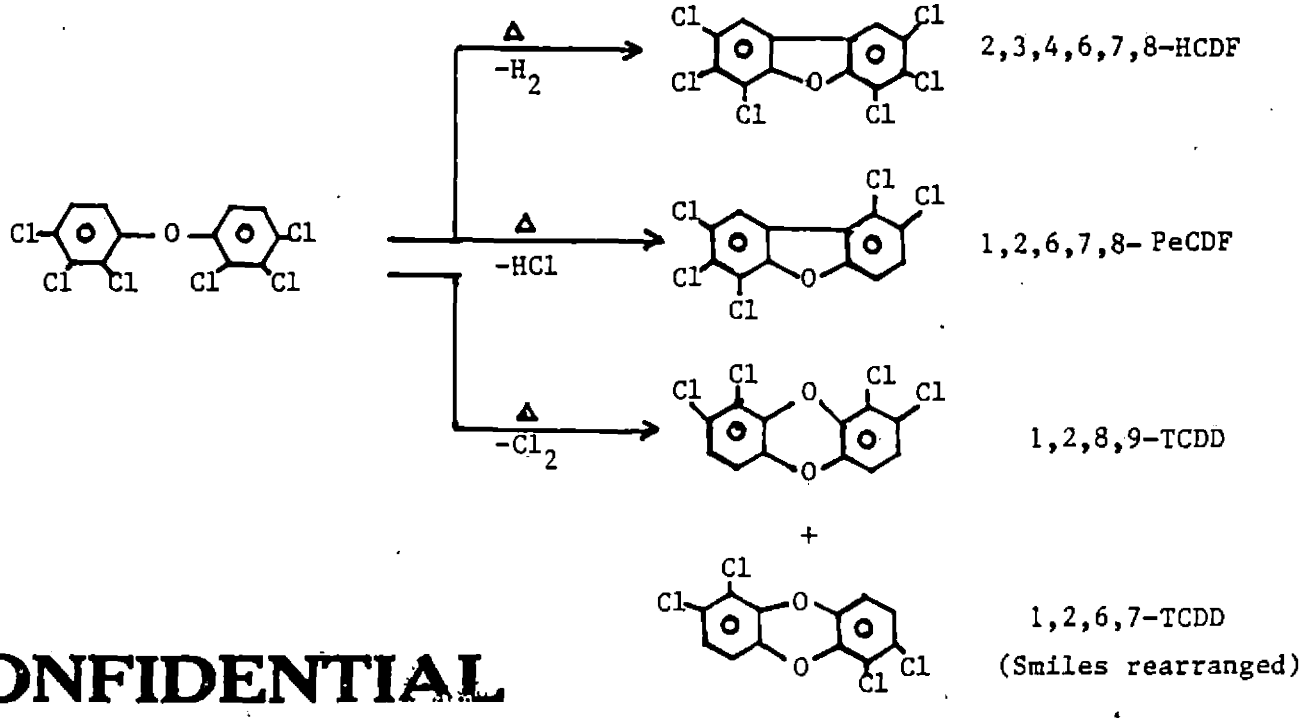
*Calculated on initial amount of I or II.

Lindahl, et al, [64] studied the laboratory pyrolysis of eight PCDPEs with three to eight chlorine atoms at 500, 600 and 700°C. No isomerization or dechlorination to lower chlorinated PCDPEs was observed. Among the decomposition products, PCDDs and PCDFs were observed with PCDFs predominating. The highest yields were obtained at 600°C. At 700°C. most of the PCDDs and PCDFs decomposed. In addition, chlorobenzenes were observed from all PCDPEs pyrolysed. Table 59 gives the results of these pyrolysis experiments.

Table 59
Pyrolysis of PCDPEs at 600°C

Compound	Decomposition %	PCDDs formed (ug/100 ug)	PCDFs formed (ug/100 ug)
3,5,4'-tri CDPE	70	4.5	-
2,4,5,4'-tetra	30	4.4	-
2,4,6,3',5'-penta	40	3.5	0.1
2,3,4,2',3',4'-hexa	50	1.4	0.8
2,3,4,2',3',5'-hexa	60	1.0	0.3
2,3,4,2',4',5'-hexa	75	0.7	0.8
2,4,5,2',4',5'-hexa	40	1.3	1.3
2,3,4,5,6,2',3',4'-octa	99.9	3.8	0.6

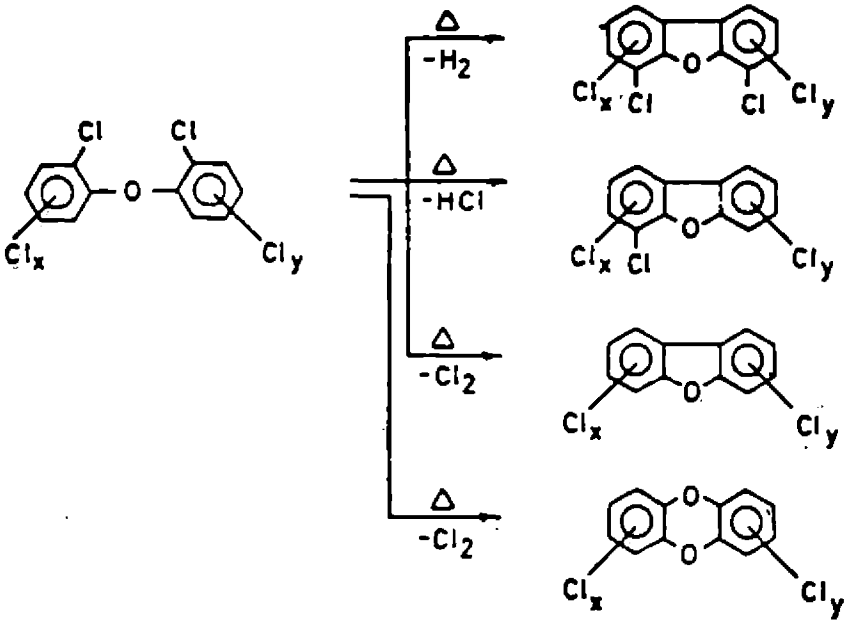
As an example of the chemistry, the pyrolysis of 2,3,4,2'3'4'-hexa CDPE gave one HCDF, one PeCDF and two TCDD isomers identified as shown below. No TCDFs were found. Minor amounts of two other



PeCDFs were observed, but the identity and source of these compounds is unknown. The 2,3,4,6,7,8-HCDF was formed by loss of ortho - H_2; the 1,2,6,7,8-PeCDF, by loss of ortho - HCl; and the 1,2,8,9- and 1,2,6,7-TCDD by the loss of ortho - Cl_2 (normal product and Smiles rearranged product, respectively).

To sum up the reaction mechanics (see Figure 3), the formation of PCDFs from the pyrolysis of PCDPEs was found to follow two main reaction routes involving the loss of ortho - H_2 and ortho - HCl. In one case (OCDPE), the formation of two PCDFs via the loss of Cl_2 (involving a rearrangement) was also observed. In addition to PCDFs, the formation of PCDDs was also observed. In this case, the formation is primarily via the loss of ortho Cl_2 with Smiles rearrangement occurring.

Figure 3
Reaction Mechanics of Formation of PCDFs and PCDDs by Pyrolysis of PCDPEs

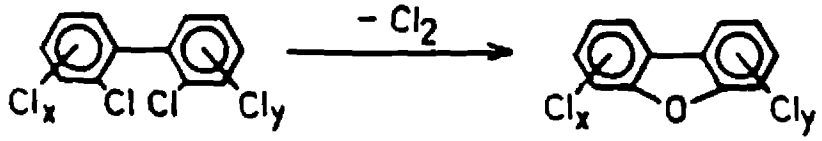


Finally, Janssens and co-workers [45] have found PCPPs and PCDPEs in emissions from a municipal incinerator in Beveren, Belgium. Since significant quantities of PCDDs and PCDFs were also found in the emissions (see Tables 23 and 24, pp. 24 and 25), this provides further evidence for PCPPs and PCDPEs being precursors of these materials in combustion processes.

many reference compounds. The researchers found the cyclization processes in these pyrolyses are intramolecular and can be described by the following 4 reaction routes leading to different PCDF isomers from the same PCB:

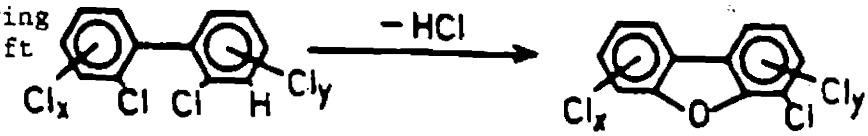
Route 1

Loss of ortho - Cl_2



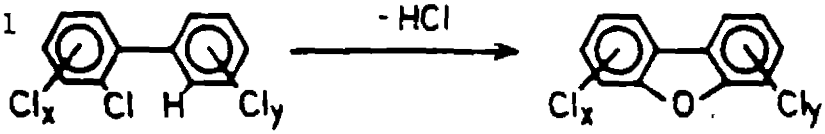
Route 2

Loss of HCl involving a 2,3-chlorine shift



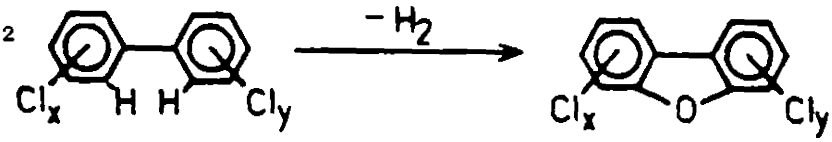
Route 3

Loss of ortho - HCl



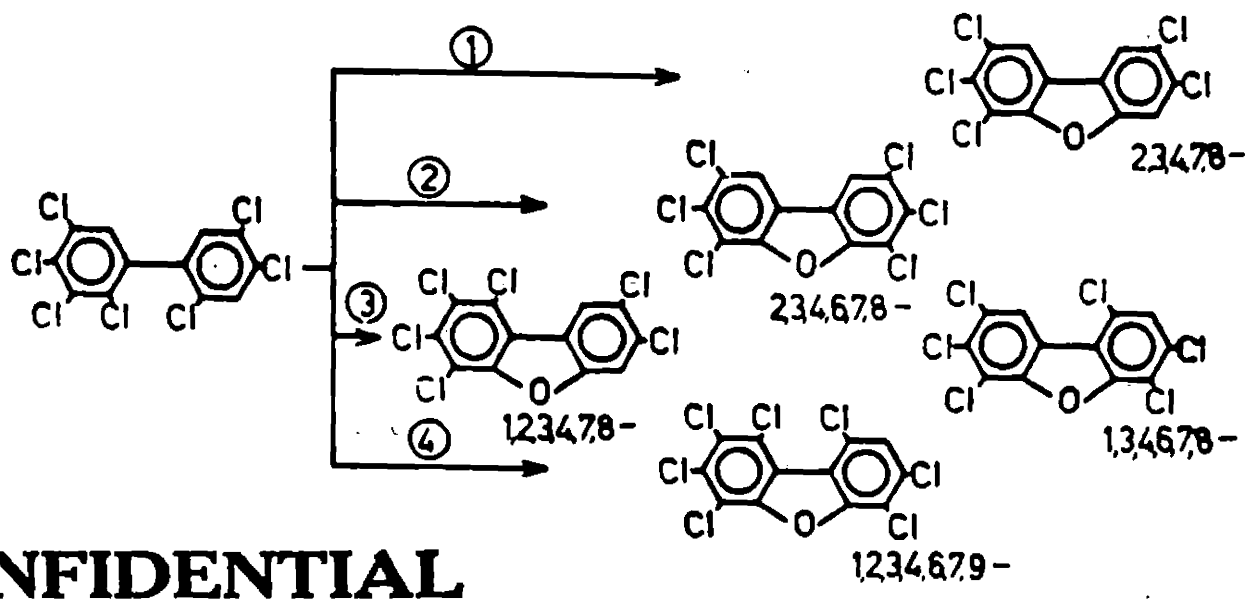
Route 4

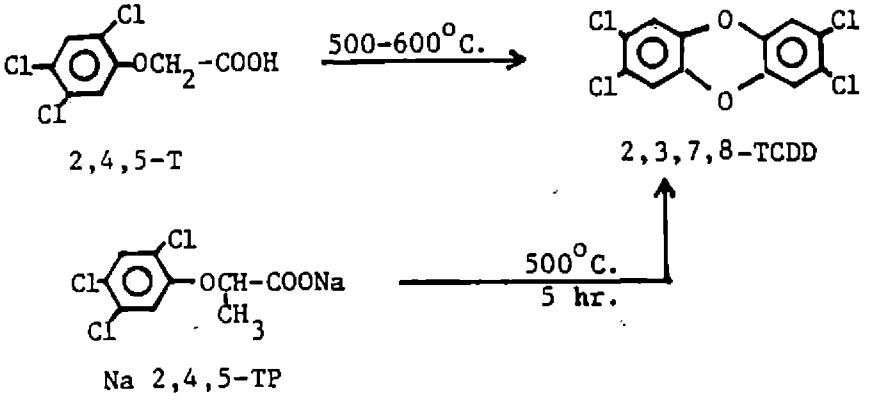
Loss of ortho - H_2



The intramolecular nature of these processes is indicated by the fact that pyrolysis of a known mixture of PCBs does not result in the formation of PCDFs other than those formed during pyrolysis of the PCBs separately [131].

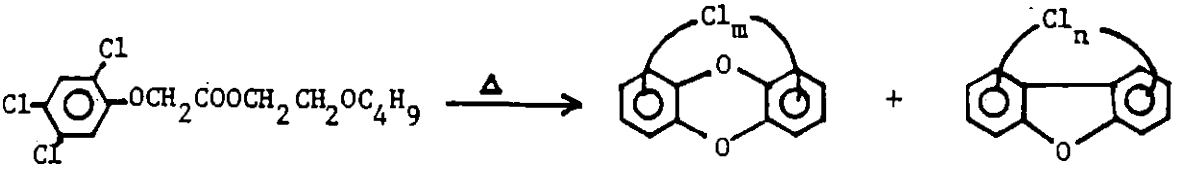
As an example, let us look at the results of the pyrolysis of 2,3,4,5,2',4',5'-heptachlorobiphenyl (HpCB) shown in the following scheme:





In 1977, Stehl and Lamparski [99] pyrolyzed grass and paper coated with 2,4,5-T, its sodium salt, and the butyl ester. All gave 2,3,7,8-TCDD in yields of 1.2×10^{-5} to 16×10^{-5}%. Grass impregnated with 2,4,5-T (equivalent to 12 lbs. per acre) gave the higher values. The purpose of this work was to find out the extent of the thermal formation of 2,3,7,8-TCDD from precursors under the conditions as "natural" as possible. Thus the burning was carried out at its own rate with good air supply.

The most comprehensive study to date was performed by Ahling, et al, [2] in 1977, who performed laboratory combustion studies on herbicide formulations containing the 2-butoxyethyl ester of 2,4,5-T. PCDDs and PCDFs were formed. The conditions in the



burning experiments together with the emitted amounts of PCDDs and PCDFs bearing 4 to 8 chlorine atoms are given in Table 61. The results show that, with few exceptions, only TCDDs are formed. This emission decreases with increased temperature and transit time. The very low temperature under "open fire" conditions (experiment 8) does not give large amounts of TCDDs. According to the researchers, the explanation is that the formation of PCDDs is

Table 61
PCDDs and PCDFs from Combustion of a Herbicide Formulation Containing 2-Butoxyethyl 2,4,5-T

Experiment no		1	2	3	4	5	6	7	8
Temperature (°C)		500	675	850	500	625	850	750	100
Transit time (s)		0.7	0.6	0.7	0.8	0.8	0.9	3.7	1.0
O_2 in flue gas (%)		15.0	12.0	10.5	13.0	12.0	7.5	0.0	17.5
CO_2 in flue gas (%)		2.6	4.0	6.1	4.1	7.4	7.7	9.0	0.3
CO in flue gas (%)		0.0	0.0	0.0	0.0	0.0	0.0	5.5	0.0
Emitted amounts (ug/kg formulation)									
Dioxins	4CDD	3.0	2.3	≤0.1	1.3	0.4	0.2	1.4	2.5
	5CDD	≤0.4	≤0.5	≤0.2	≤0.6	≤0.3	≤0.3	1.2	≤0.5
	6CDD	2.0	≤0.5	≤0.2	≤0.6	≤0.3	≤0.3	0.8	≤0.5
	7CDD	≤0.4	≤0.5	≤0.2	≤0.6	≤0.3	≤0.3	0.3	≤0.5
	8CDD	≤0.4	≤0.5	≤0.2	≤0.6	≤0.3	1.4	0.05	≤0.5
Furans	4CDF	≤0.2	8.0	1.3	0.6	0.2	2.9	7.6	1.0
	5CDF	1.8	7.1	1.9	1.3	≤0.3	4.2	5.7	0.5
	6CDF	1.3	2.8	1.8	≤0.6	≤0.3	4.9	1.7	≤0.4
	7CDF	≤0.7	≤0.7	1.4	≤0.6	≤0.3	3.4	≤0.2	2.3
	8CDF	≤0.7	≤0.7	≤0.6	≤0.6	≤0.3	3.9	≤0.1	≤0.4

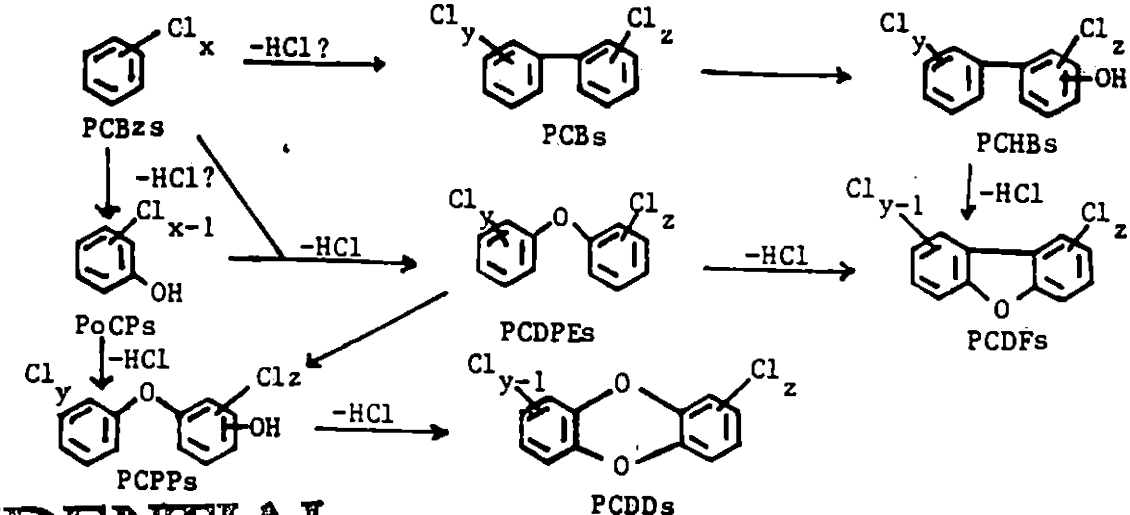
insufficient at this low temperature or that the formed PCDDs are effectively destroyed during the relatively long transit time. No explanation for the formation of the higher chlorinated PCDDs in some cases, especially under oxygen deficient conditions in experiment 7, could be given by the researchers. Background levels of PCDFs were unexpectedly high. The PCDF results in Table 61 are not corrected for these background levels, thus the low values must be considered questionable. In most experiments, the PCDF emissions were higher than those of the PCDDs. These amounts of PCDFs cannot be explained by the formulation contents. Also the emissions of PCDFs can not be correlated with temperature or transit time, whereas the PCDD emissions decrease with increased temperature and transit time. Based on other studies, the minor amounts of chlorophenols (2.42×10^{-4} g/g) and PCDDs ($\sim 20 \times 10^{-9}$ g/g) present in the formulation cannot give the amount of PCDDs found. The 2,4,5-T impurity (4.7×10^{-2} g/g) could possibly have an impact on the results serving as a precursor. The largest formation of TCDDs found in this study corresponds to a formation of about 1 ug per m^2 in a forest fire.

f. A Summation of Precursor Chemistry and Other Related Laboratory Experiments

In 1982, Janssens and co-workers published an outstanding article [45] . The focus of the experimentation was a systematic monitoring of the PCDDs and PCDFs and their possible precursors emitted by a municipal incinerator in Beveren, Belgium. One of the major purposes of the study was the definition of the role of the PCBzs and PoCPs as precursors for the PCDDs and PCDFs in the actual conditions of the waste burning process. The sampling of the particulate phase and the vapor phase of the emissions, the extractions, and the purification techniques for the various chlorinated compound types were state-of-the-art. Analyses of the PCDDs and PCDFs were performed by HRGC/MS with EI source with quantitation done by the MID technique. Table 23 (p.24) summarizes the whole of the emission data (particulate phase + vapor phase). The findings were in agreement with data from other investigators. Also several PCDPEs and PCPPs as well as some PCBs were found, but only as minor constituents in the flue gas.

The researchers pointed out that only a minor fraction of all of the compound types (< 10% in most cases) is found on the particulates. Thus analysis of fly ash samples is in no way relevant for the total emission.

Of real significance, the authors postulated a pyrosynthesis mechanism based on data from the many laboratory studies performed by other researchers on the pyrolysis of possible precursors, which we have discussed. They claim that this mechanism is confirmed by their identification of the above mentioned intermediates as well as by the correlation of the concentration levels of the precursors with those of the PCDDs and PCDFs in the actual conditions of refuse burning. Their mechanistic scheme for the formation of PCDDs and PCDFs is shown below.



The essential role of PoCPs and PCBzs as precursors is obvious. This study does seem to provide strong experimental evidence for the proposed overall mechanics. Janssen and co-workers also showed PVC going to PCBzs. We omitted this reaction step, because we consider such a conversion as definitely part of the "de novo" synthesis mechanics, which will be discussed in the next section. The authors suggest such a possibility in their discussion of the experimental results.

Related to this precursor mechanistic approach, laboratory studies by Olie, et al, [77] on the burning of lignin sulfonate (a polyphenolic) in the presence of HCl or PVC (a chlorine source) to give PCDDs and PCDFs tend to support the Janssen scheme. Also, Liberti, et al,[60,63] performed laboratory combustion experiments on vegetable materials (chestnut extract, fresh fruits and vegetables, tannic acid and mimosa extract), per se, and in the presence of chlorine in the air stream or added PVC. In the per se case, specific phenols (phenol, m-cresol and p-cresol) were formed. In the case where a chlorine donor was added, PCDDs and PCDFs were found as well as di-, tri-, tetra- and pentachlorophenols. Their mechanistic model is simple:

Precursor A (organic matrix) Phenols or polyphenols + Precursor B (Cl_2 or HCl donor) PVC $\longrightarrow$ PCDDs + PCDFs

3. PCDDs and PCDFs by "de novo" syntheses

In 1980, Lustenhouwer, et al, [84] published the first review article dealing with some possible mechanisms for thermal generation of PCDDs and PCDFs from a theoretical viewpoint. According to these researchers, very little direct evidence existed at that time for the "de novo" formation of these or related compounds from carbon sources and natural chlorine donors. The presence of PCDDs and PCDFs from pyrolysis or incineration processes without sources of anthropogenic (man-made) chlorine compounds (Dow's "Trace Chemistries of Fire") and chlorobenzenes in fly ash from coal fired installations suggest that this process is operating.

Chlorine does occur at low levels in most fuels and material used for combustion, as Table 62 shows.

Table 62

Chlorine Content of Fuels and Combustible Material

Fuel	Chlorine(ppm)
coal	1300
refuse	2500
paper	300-1600
leaded gasoline	300
unleaded gasoline	1-6

Lustenhouwer and co-workers indicated that the scarcity of data in this area does not mean that this mechanism is not operating, but only indicates that not enough work has been carried out. This seems to have spurred researchers into action.

In 1982, Janssens and co-workers [45] cited some evidence that this mechanism may be operating in their study of chlorinated organics in emissions from a municipal incinerator. They found that the contribution of the individual PCBzs to the total PCBz emission was nearly constant, and was not influenced by the concentration levels of the individual PCBzs in the flue gas or by the composition of the treated refuse. They indicated that this strongly suggests the occurrence of an alternate formation process (to their proposed "precursor" scheme) for PCDDs and PCDFs, similar to that for polynuclear aromatic hydrocarbons (PAHs), which has been studied extensively. This appears to involve the recombination of small initial radical fragments (usually containing two carbons). The different analogs are formed in characteristic concentration ratios, depending on the combustion temperature and, to some extent, on the composition of the material burned.

In view of the above findings, the authors attempted to relate the PCBz formation to the content of the organic chlorinated compounds of the burned material. Several analyses were performed on fly ash samples, obtained from wood, coal, and heating oil. The results indicate that the chemical nature of the chlorine containing compounds in the substance to be incinerated is only of minor importance for the formation of PCBzs, which again suggests an alternate mechanism.

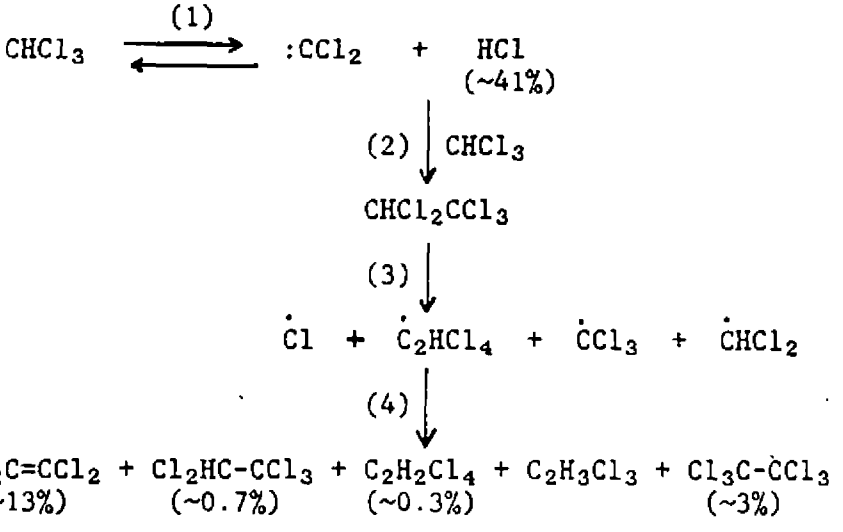
Such a suggestion sounds very much like the "Trace Chemistries of Fire" hypothesis or "de novo" synthesis. It could be that both the "precursor" scheme proposed by Janssens and co-workers and the "de novo" mechanics are operating, either independently or concerted - more likely concerted.

Also, in 1982, Choudhry, Olie, and Hutzinger [109] published an outstanding review of the literature dealing with experimentation which provides substantial indirect evidence for the "de novo" syntheses of PCDDs, PCDFs and related compounds, and the possible mechanisms involved. Like Lustenhouwer, they say that little experimental evidence is available to show thermal synthesis of chloroaromatic compounds from non-chlorinated aliphatic precursors and inorganic chlorine. This review highlights particular points of a far more detailed treatment of the "Mechanistic Aspects of the Thermal Formation of Halogenated Organic Compounds Including Dibenzo-p-dioxins", which was published in 5 parts by Choudhry and Hutzinger [18,30,85,86,127].

They divided their review into three parts. The first part deals with the thermochemical decomposition of chlorinated monomers (aliphatics and aromatics) and polymers, such as polyvinyl chloride (PVC) and lignin as well as the thermal production of PCDDs and PCDFs. In the second part, the thermolysis of organic matter is discussed along with incorporation of chlorine from inorganic chlorides with the formation of chloroorganic compounds, such as methyl chloride, carbon tetrachloride and hexachlorobenzene (HCB). In the final part, hypotheses about the thermal formation of PCDDs, PCDFs and related compounds in incinerators are formulated based on well known experimental literature. A capsule summary with pertinent examples follows.

a. Thermochemical decomposition

In aliphatic hydrocarbon chemistry, certain reactions appear to play a role in the formation of PCDDs and PCDFs. The pyrolysis of chloroform has been shown to yield the following products: [109, p. 277]

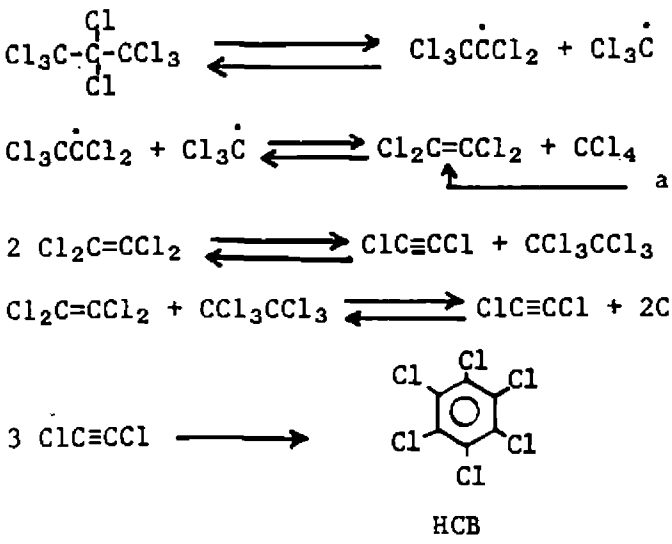


Notice that tetrachloroethylene ($Cl_2C=CCl_2$) is formed in relatively high yield.

Also, by heating any perchlorocarbon compound or mixture of compounds having an overall mole ratio of chlorine to carbon between 1 and 4, an equilibrium is reached between hexachlorobenzene (HCB)(C_6Cl_6), carbon tetrachloride (CCl_4) and hexachloroethane (C_2Cl_6). [18, p. 23] Related to this,

$$9\ C_2Cl_6 \rightleftharpoons C_6Cl_6 + 12\ CCl_4$$

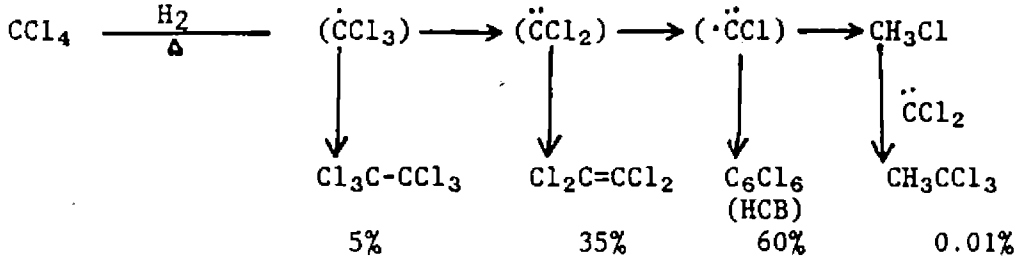
the results of the kinetic study of the pyrolysis of octachloropropane (a perchlorocarbon compound) at 350°C. shows that the following reactions occur. [18 p. 27]



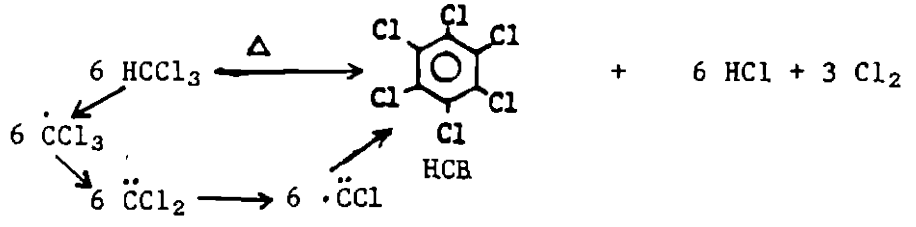
HCB

Notice that tetrachloroethylene ($Cl_2C=CCl_2$) and dichloroacetylene ($ClC\equiv CCl$) are intermediates in this sequence of reactions, and that the end result is HCB (a PCBz). It has been known for a long time that dichloroethylene even at room temperature will form HCB. [18 p. 27] This supports the intermediate nature of dichloroacetylene. Very recently, the decomposition of acetylene has been conclusively proven to be primarily responsible for the growth of soot particles in combustion processes [145].

The reaction of carbon tetrachloride (CCl_4) and hydrogen in a hot tube at 600-650°C. gives the following products [18 p.29].

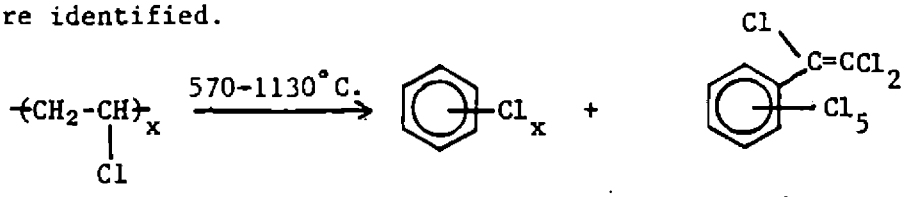


Notice the formation of HCB in high yield from the chlorocarbene radical. Also, it is known that the pyrolysis of chloroform yields HCB, HCl and Cl_2. [18 p.30] Here again the obvious intermediate seems to be the chlorocarbene radical ($\cdot\ddot{C}Cl$).



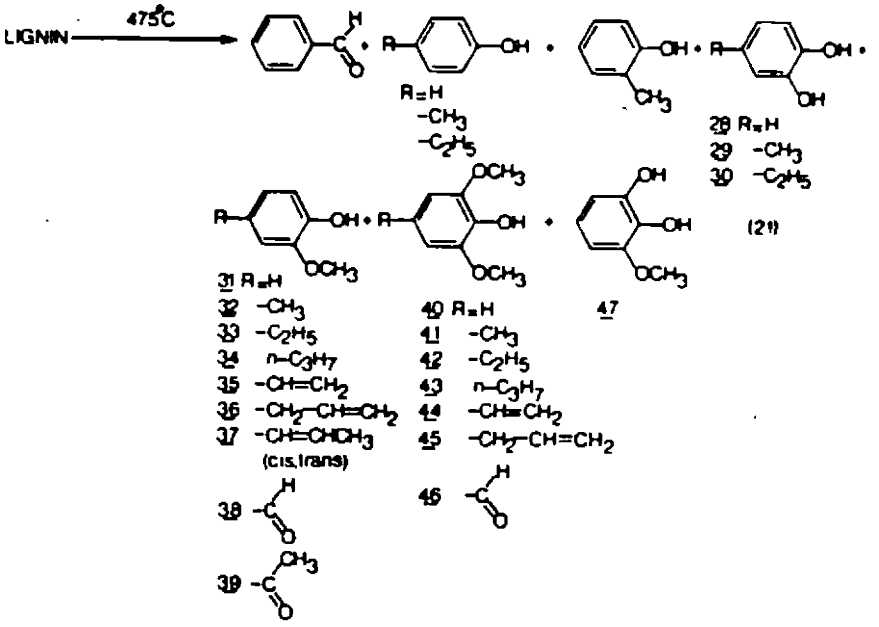
Concerning synthetic aliphatic hydrocarbon polymers, a large number of these materials are commonly used in daily life. The major commercial polymers include plastics, such as polyvinyl chloride (PVC), polyethylene and polyesters. PVC is one of the three most important plastics in use. Some

of its applications are cable insulation, leathercloth (car seat covers), packaging, and toys. Many researchers have studied the pyrolysis of PVC. Of particular significance was the work by Ahling and co-workers. [109,132] Eleven pyrolysis experiments were performed at temperatures between 570-1130°C. at different oxygen levels. Chlorinated benzenes (PCBzs) from dichloro- to hexachlorobenzenes including several isomers of dichloro-, trichloro-, and tetrachlorobenzene were identified. In addition, PCBs and octachlorostyrene (OCS) were identified.



Notice the trichloroethylene moiety (a two carbon radical) present in OCS. From our discussion of possible precursors to PCDDs and PCDFs, PCBzs and PCBs appear to be two of them.

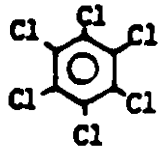
Concerning natural polymers, lignin is an extremely complex one. It is biosynthesized by plants and it is the second most abundant organic material in the environment. It is classified as a polyphenolic. Pyrolysis of this material at 475°C. resulted in the formation of 26 compounds as shown below [109 P·291]. All of the phenols generated could certainly represent precursors for PCDDs and PCDFs if a chlorine source were present.



You will recall, in the discussion of the chemistry of PCDD precursors (page 79), the laboratory studies by Olie, et al, [77] on the burning of lignin sulfonate in the presence of HCl or PVC to give PCDDs and PCDFs, and the work of Liberti [60, 63] on the combustion of vegetable materials in the presence of Cl_2 or added PVC to give polychlorinated phenols (PoCPs) as well as PCDDs and PCDFs. Similar studies by Ahling and Lindskog [110] on the pyrolysis of concentrated bleaching effluents from wood pulp (contains chlorinated degradation products of lignins) and wood pulp black liquor (prior to bleaching), which contains ~ 6g/liter of inorganic chlorine but no organic chlorine compounds, gave PCBzs. The formation of PCBzs was greatest from the pyrolysis of black liquor.

b. Incorporation of inorganic chlorine into an organic molecule

We have already discussed the studies by Ahling and Lindskog [110] on the pyrolysis of black liquor to form PCBzs and by Mahle and Whiting [67] on the combustion of bituminous coal in the presence of NaCl, HCl and Cl_2 resulting in PCDDs (see Table 43, p. 42). Also petroleum coke has been chlorinated and pyrolyzed in a potassium chloride, sodium chloride melt. [109 p 292]. The following equation shows the results. Table 63 gives the results of the coke pyrolysis

Petroleum coke $\xrightarrow[Cl_2,\ KCl/NaCl]{700°C.}$ CCl_4 +  + HCl + $COCl_2$ + CO

HCB

+ CO_2 + H_2S + H_2 + CH_4

at various temperatures. The formation of the chlorocarbons

Table 63

Chlorination and Pyrolysis[a] of Coke[b]

in a KCl/NaCl Melt[c]

Temperature (°C)	Products (mg/g)			
	CCl_4		C_6Cl_6	
	2h[d]	4h[d]	2h[d]	4h[d]
700	2.0	2.3	0.5	0.7
800	1.5	1.5	0.0	0.0
900	0.1	0.1	0.0	0.0
1000	0.0	0.0	0.0	0.0

[a] During the process, the helium flow (free of oxygen traces) and dried chlorine (100%) flow were 2 and 7.5 l/hr, respectively. In all the experiments, 80 gm of the melt and 1 gm of petroleum coke were utilized

[b] The composition (wt. %) was: H, 3.51; C, 94.5; ash, 0.5; S, 0.85; and (O + N), 1.2

[c] The molecular ratio of the melt was one to one

[d] Time of pyrolysis

is believed to be the result of interaction of Cl_2 with the irregular side structure of the coke macromolecules. At temperatures of 900-1000°C., the decomposition of the side structure probably takes place faster than the direct chlorination, thus no chloroorganics are formed at ~ 1000°C.

Another interesting study involves the combustion of tobacco. [109,p. 294] When tobacco is burned methyl chloride (CH_3Cl) is formed. The methyl chloride can come either from the organic or inorganic chlorine present in the tobacco (~0.25% and ~0.88%, respectively). When tobacco impregnated with radioactive inorganic chloride was burned, radioactive methyl chloride ($CH_3{}^{36}Cl$) was formed.

$$\text{Tobacco} + {}^{36}Cl^- \xrightarrow{\text{smoking}} CH_3{}^{36}Cl$$

Concerning the formation of molecular chlorine (Cl_2) or atomic chlorine ($\dot{C}l$) necessary for chlorination of an organic molecule, the formation of chlorine from salt, sand and water has been known for many years. The following reactions are involved at temperatures of 600-1000°C. [85,p. 280] The first reaction occurs in dry air, whereas all three occur in moist air, with the second reaction

$$\underset{\text{salt}}{4NaCl} + \underset{\text{sand}}{SiO_2} + O_2 \xrightarrow{\Delta} 2Na_2O \cdot SiO_2 + 2Cl_2$$

$$2NaCl + SiO_2 + H_2O \xrightarrow{\Delta} Na_2O \cdot SiO_2 + 2HCl$$

$$4HCl + O_2 \rightleftharpoons 2H_2O + 2Cl_2$$

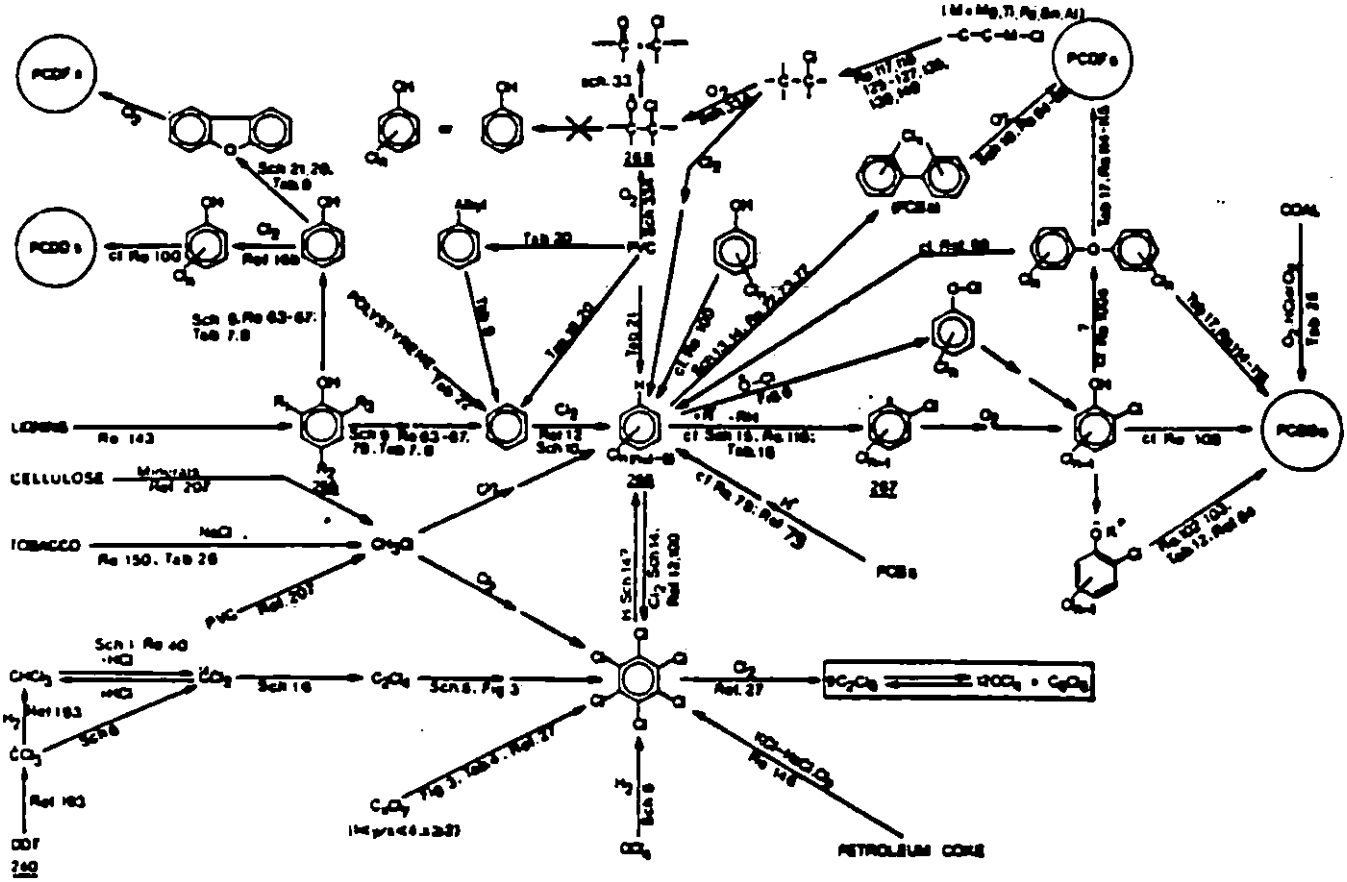
predominating. The last reaction is the basis for several industrial chlorination processes.

c. Hypotheses about the thermal generation of PCDDs, PCDFs, and related compounds in incinerators ("de novo" syntheses)

After reviewing all of the literature, Choudhry, et al, consolidated the information into a reaction flow chart to illustrate many of the hypothetical possibilities for the "de novo" syntheses of PCDDs, PCDFs, and related compounds. These are shown in Figure 4. [86,109]

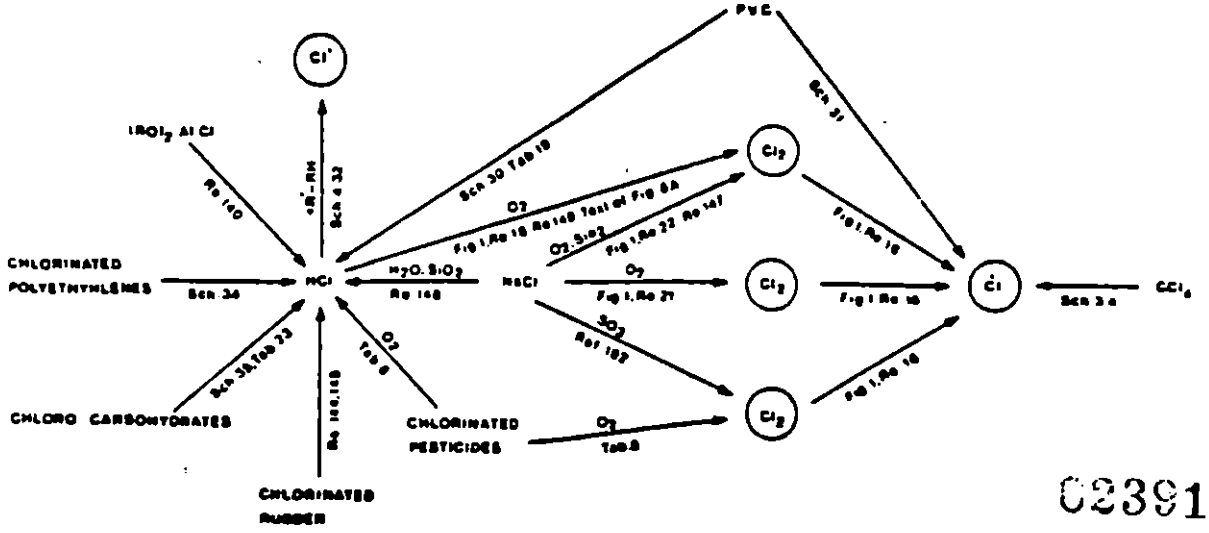
Figure 4

Complex Array of PCDD and PCDF Syntheses from Chemically Unrelated Organic Matter ("de novo" Syntheses)



They also published another reaction flow chart on some possible sources of molecular and atomic chlorine (Cl_2 and $\dot{C}l$) in incinerators [86, P·303]. This is shown in Figure 5.

Figure 5
Possible Sources of Cl_2 and $\dot{C}l$



Within the past year, Ballschmiter and co-workers [5] published preliminary results of their study on the emissions from a municipal incinerator. Their goal is to correlate the operating conditions of an incinerator with the organics found in the particulate emissions, primarily PCDDs and PCDFs. As yet, they have found no correlation of the PCDD content in the fly ash with other measured parameters. They compared HCl, SO_2, CO_2, H_2O, O_2 emissions in the stack gases with PCDD formation. They were particularly interested in HCl where there appears to be no correlation at all. Comparing the sum of bond dissociation energies of reactions involving the reactants HCl and chlorine, thermodynamics seem to back these experimental results, according to the authors.

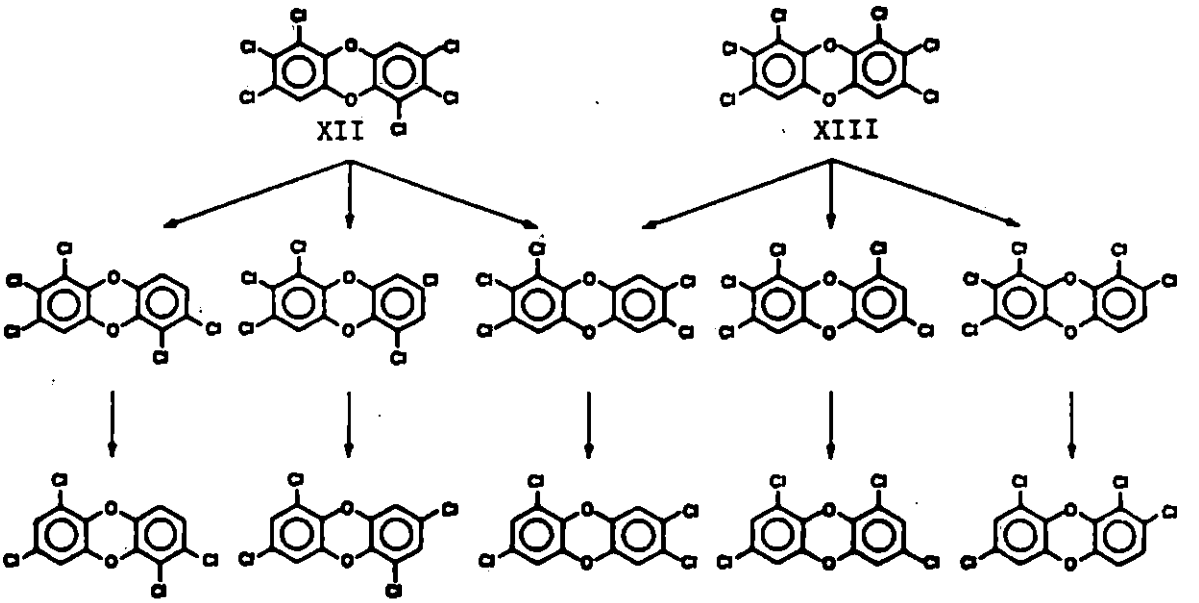
The authors obviously are advocates of the "de novo" synthesis hypothesis, since their introduction in this publication discusses the chemistry in flames being the chemistry of radicals in complex reaction pathways. They suggest that the chemistry in flames of incinerators, due to the variation in input and operating conditions, will be an extreme in complexity and can be dealt with only in very general terms. In addition to oxidation as the main type of reaction in a non-reductive flame, addition elimination, reduction and chlorination will be further reaction pathways, at least as intermediate routes. Products formed will be the object of pyrolytic reactions in the gas phase as well as in the adsorbed state on particles. Metal-catalyzed reactions on particles also must be considered. The chemistry in flames, they say, is governed by formation of stable radicals and olefinic and acetylenic compounds. They indicate that chloroethenes (chloroethylenes) and chloroethynes (chloroacetylenes) appear to play a central role as intermediates in the formation of PCDDs, PCDFs, and other chlorinated aromatic compounds. They also published a series of theoretical radical reactions that possibly occur in an incinerator. These reactions involve radicals of the C=C, C=O, C=N, O, OOH and Cl type.

From the results obtained thus far, these researchers believe that municipal incinerators can in practice be run under conditions of very low level of emissions of organic compounds. The authors did not elaborate on this point.

Back to Choudhry, et al, and their mechanistic review of the thermal formation of PCDDs and PCDFs, [109] they conclude that there is no doubt that "de novo" syntheses play a significant role in incinerators and other fires. They say it is evident that the PCBzs and PoCPs are key intermediate compounds of prime importance in the "de novo" synthesis of PCDDs and PCDFs from any organic matter. In other words, both Choudhry, Olie and Hutzinger as well as Ballschmiter and his co-workers support Dow's "Trace Chemistries of Fire" hypothesis. After digesting all of the literature to this point in time, there is no question in our minds that "de novo" syntheses (chemistry in flames, "Trace Chemistries of Fire" - call it what you may) play a significant role in the formation of PCDDs and PCDFs in incinerators, probably in conjunction with ordinary chemical reactions involving the described "precursor" chemistry.

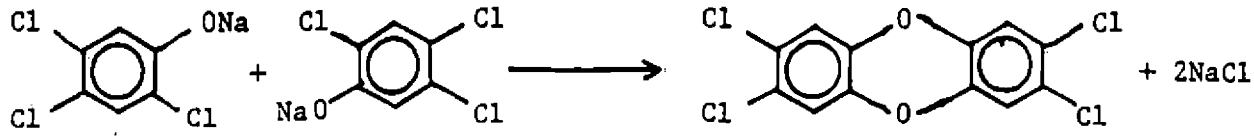
of PeCDDs and TCDDs from two isomeric HCDDs, 1,2,3,6,7,8- and 1,2,3,7,8,9-HCDD (XII and XIII, respectivly) again from photolysis in benzene-hexane (5:95 by vol.) solution (see Figure 6). All photoproducts were identified by comparing retention times and mass

Figure 6
Photolysis of 1,2,3,6,7,8- (XII) and 1,2,3,7,8,9-HCDD (XIII) in Benzene/n-Hexane (5:95 by vol.) Solution



spectra with those of reference compounds. The formation of 1,3,6,8- and 1,3,7,9-TCDD as the principal isomers from the photolysis of 1,2,3,6,7,8- and 1,2,3,7,8,9-TCDD, respectively, clearly shows a preferential dechlorination from the lateral positions (2,3,7 and 8 positions) as opposed to dechlorination from the peri positions (1,4,6 and 9 positions). Dobbs and Grant [114] observed another pertinent fact. Compounds possessing the same number of chlorine atoms in the 2-positions (lateral positions) photolyse more rapidly ($T_{\frac{1}{2}}$ is shorter) as chlorine atoms are removed from the 1-positions (peri positions). Extrapolation of these results led them to predict that 2,3,7,8-TCDD should be the most photolabile of the PCDDs. According to Dobbs and Grant, their observations are reassuring from the environmental viewpoint that the PCDDs most likely to be the most toxic are also the most susceptible to photodegradation, and the photodegradation of PCDDs is not a facile route for the generation of highly toxic PCDDs.

Nestrick and co-workers [113, p.144] studied the photodegradation of all 22 TCDD isomers. Table 66 summarizes the data for dilute n-hexadecane solutions and for material exposed on a clean, soft-glass surface. These data show that 2,3,7,8-TCDD is a very unusual isomer with respect to photolytic behavior. It has the



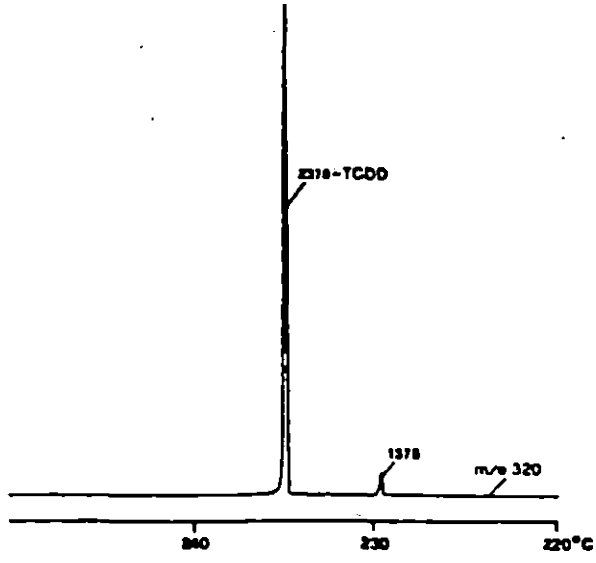
NaTCP

2,3,7,8-TCDD

overchlorinated benzenes in the 1,2,4,5-TCB, the sodium salts of di- and tetrachlorophenols present in the NaTCP might produce trace quantities of lower or higher chlorinated PCDDs.

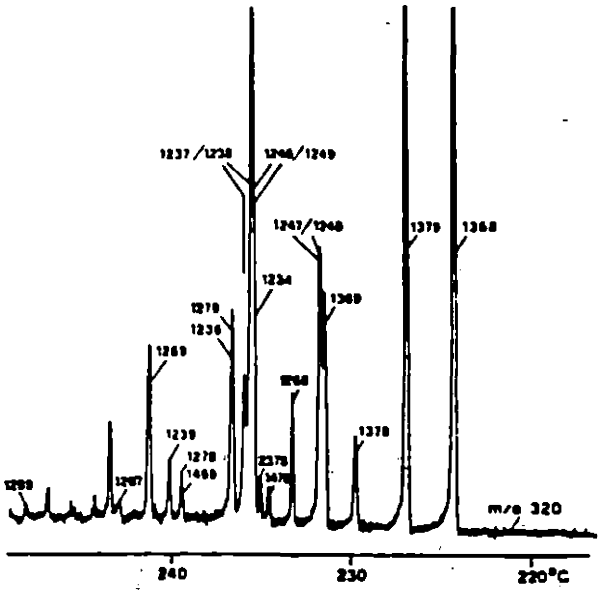
In 1976, an explosion associated with the production of NaTCP at the ICMESA plant at Seveso, Italy sprayed the reactor contents over a densely populated area. Realizing that considerable 2,3,7,8-TCDD would likely be present, Buser and Rappe [140], in 1980, analysed soil samples from the contaminated area in Seveso for TCDDs. They used a HRGC/MS technique capable of separating and identifying all 22 possible TCDD isomers. Figure 7 shows the mass spectroscopic analysis of the soil extract. Notice that only two TCDD isomers are present, with the

Figure 7
Selected Ion Chromatogram of Seveso Soil Extract Showing TCDDs Present



2,3,7,8-TCDD overwhelmingly dominant. For comparison, the researchers analysed the extract of a fly ash sample from a municipal incinerator in Switzerland using the same technique. Figure 8 shows the mass spectroscopic analysis of the fly ash extract. The chromatogram shows a complex isomeric mixture

Figure 8
Selected Ion Chromatogram of Fly Ash
Extract Showing TCDDs Present



with at least 17 of the possible 22 TCDD isomers present. The major isomers are 1,3,6,8-, 1,3,7,9- and 1,2,3,7-(or 1,2,3,8-) TCDD, which make up more than half of the total TCDD amount present. Notice that the 2,3,7,8-TCDD isomer is present at a very low level (~1% of the total TCDD amount). The authors said that this same isomeric pattern was observed in other fly ash extract samples. Since only the mass/charge ratio (m/e) of 320 was measured, higher chlorine number homologs are not shown in Figures 7 and 8.

Another example of the limited number of PCDD congeners generated by industrial processes is the production of 2,4-D and its formulated amine salts and esters. 2,4-D is produced by the condensation of 2,4-dichlorophenol (2,4-DCP) with monochloroacetic acid under basic aqueous conditions. The 2,4-dichlorophenol is produced by the chlorination of phenol. The commercial material usually contains some 2,6-dichlorophenol as well as 2,4,6-trichlorophenol as impurities.

Table 67
Di-, Tri- and Tetra CDDs in 2,4-D Amine Formulations

Sample No.	Ingredient label guarantee (oz. 2,4-D amine per gallon)	2,4-D acid % (w/w) equiv.	Dioxin* (ppb) 2,7-di-	1,3,7-tri-	1,3,6,8-/1,3,7,9-tetra-
1	80	44.6	–	–	–
2	80	–	–	–	–
3	80	–	–	–	–
4	80	41.3	–	–	–
5	80	–	–	–	–
6	80	41.7	–	–	–
7	80	37.9	–	–	–
8	80	–	–	–	–
9	80	42.44	316	490	132
10	80	43.10	275	587	136
11	80	–	–	–	–
12	80	–	–	–	–
13	80	42.9	409	551	210
14	80	42.9	–	–	–
15	80	–	–	–	–
16	80	–	–	–	–
17	80	–	–	–	–
18	80	–	–	–	
19	80	42.55	140	230	96
20	80	42.66	–	38	54
21	80	42.74	–	584	278
22	80	42.74	5	54	20
23	96	49.59	33	533	208
24	80	42.74	–	–	–
25	80	–	–	–	–
26	80	–	–	–	–

* All dioxin results are based on 2,4-D acid % (w/w) equiv.

– Indicates none detected above 1 ppb.

the isooctyl ester (IOE), mixed butyl ester (MBE) and propylene glycol butyl ester (PGBE) types. The PCDD levels ranged from 104 ppb to 23.8 ppm for 2,7-DCDD, 35 ppb to 2.45 ppm for 1,3,7-TrCDD, and 120 ppb to 8.7 ppm for 1,3,6,8-/1,3,7,9-TCDD. No other PCDD congeners were found in these samples.

HPLC analysis of technical grade 2,4-DCP showed that, in addition to 2,4-DCP (86.22%), it contained 2,6-DCP (5.64%), 2,4,6-TCP (3.23%) and o- and p-chlorophenols (1.85%). The

ST. LOUIS POST-DISPATCH Tues., Apr. 10, 1984

Municipal Incinerators Appear To Be Spouting Dioxin, Swedish Researcher Says

A Swedish researcher who found dioxin in mother's milk and in human fat tissue says the discovery shows that municipal incinerators are contaminating the environment with the toxic chemical.

Christoffer Rappe, of the department of organic chemistry at the University of Umea in Sweden, reported his findings Monday in St. Louis at the national meeting of the American Chemical Society.

Rappe said that 2,3,7,8-TCDD had been found in all the samples in his study. That type of dioxin is the same found at Times Beach and at 36 other sites in Missouri. Seven samples of mother's milk and 25 samples of fat tissue were collected at random in West Germany and northern Sweden.

Tests showed that the levels of dioxin in the samples had ranged from about 4 parts of dioxin for each trillion parts of fat or milk to about 40 parts for each trillion, Rappe said. He said this was not a dangerous level, but because dioxin can accumulate in the body, continued exposure to incinerators that produce the chemical could prove to be harmful.

Rappe said he did not know how much the subjects in the study had been exposed to incinerator pollution. But he said that the types of dioxin found in the samples included some produced only through incineration, indicating that the more dangerous type of dioxin also came from the same source.

Asked what could be done to prevent problems, Rappe replied: "Construct better incinerators."

He said an incinerator of the type being considered in an experiment to burn dioxin-contaminated soil in Missouri would present no problems because it presumably would have pollution-control equipment to prevent the spread of dioxin.

European scientists have been concerned for several years by tests showing dioxin, dibenzofuran and heavy metals coming from municipal incinerators. The scientists are uncertain about why the toxic chemicals are formed. Some speculate that the dioxin is formed by burning plastics containing chlorine.

B. Atmospheric and Surface Photodegradation

As mentioned in the Photochemistry discussion in the Chemistry section (p. 88), photodegradation of PCDDs and PCDFs in the atmosphere (gas phase) is believed to occur but there is very little information available. It has been reported [66] that a "half-life" for 2,3,7,8-TCDD in the gas phase under environmental sunlight conditions would be on the order of 5 to 24 days. In contrast, the same researchers suggest that oxidation of 2,3,7,8-TCDD by the hydroxyl radical in the atmosphere would be rapid.

Mill [137] indicates that the low vapor pressure of 2,3,7,8-TCDD suggests that some of the PCDDs emitted to the atmosphere from incinerator sources will adsorb to particulate matter and either remain in suspended particulate or return to the soil and surface waters. That fraction of PCDDs remaining in the vapor phase, Mill says, could undergo photochemical degradation in the same way as 2,3,7,8-TCDD dissolved in water. He says kinetic rate laws governing photolysis of

chemicals in the atmosphere are virtually identical to those for photolysis of dilute aqueous solutions, and the photolytic "half-life" for 2,3,7,8-TCDD vapor in summer sunlight could be as low as ½ hour.

More than likely, the majority of the PCDDs emitted to the atmosphere will be strongly adsorbed to particulate matter, and the photolysis rate may be quite different than that in the gas phase. Townsend [112] has investigated, theoretically, the fate of PCDDs in the atmosphere in terms of the change in isomer distribution at various atmospheric sites. His results suggest that airborn microparticulates transport and mix the lower volatility CDDs, and that photodegradation may be a major loss process for them.

Janssens, et al, [45] also discusses the adsorption of PCDDs on particulate matter in the atmosphere. It can be expected that during the cooling of the flue gases in the atmosphere, the PCDDs and PCDFs in the gas phase will probably adsorb rapidly and almost quantitatively onto the particulate matter, especially the very small, respirable particles. This is especially true for the less volatile PCDDs and PCDFs. These researchers indicate that further investigations of ambient aerosols are necessary.

Mill [137] goes on to say that the major transformation process in the environment for most organic chemicals is oxidation by the OH radical. Using chlorine and phenoxy substituents, he estimated the "half-life" for 2,3,7,8-TCDD in the atmospheric vapor phase to be 319 hours. He says we have no data to evaluate the rate of oxidation of 2,3,7,8-TCDD adsorbed to atmospheric particulate matter, but suspects that the rate will be much slower. Thus, according to Mill, these estimates show that photolysis is the dominant process in the atmospheric vapor phase. In our opinion, this is awfully thin data and there is a definite need for gas phase and microparticulate adsorbed phase photodecomposition rate studies on PCDDs and PCDFs, together with the determination of the photolytic products. Such studies can have great significance with regard to the risk of human exposure.

Data on the photolytic decomposition of PCDDs on solid surfaces is also limited. In contrast to the rapid photodecomposition of 2,3,7,8-TCDD in organic solvents, Crosby, et al, [144] irradiated this compound on dry as well as wet soil (sandy or silty clay loam soil) supported on a glass plate for 96 hours, and on the surface of a glass plate as a thin dry film for 14 days. No observable photodecomposition occurred. Methanol was used as the carrier solvent for the preparation of samples of 2,3,7,8-TCDD adsorbed on solid surfaces for these photolytic experiments. No photodecomposition in the soil experiments was probably due to the fact that when the methanol solution of 2,3,7,8-TCDD was applied to the soil, it soaked in and deposited much of the 2,3,7,8-TCDD within the soil where light could not reach it. Also another reason for no photodecomposition of 2,3,7,8-TCDD in all of these cases is

VII. ANALYTICAL TECHNIQUES

Analysis of PCDD and PCDF emissions from combustion sources is a very sophisticated operation for several reasons. First of all, we are dealing with trace quantities of materials usually in the ppb or ppt range. Secondly, many organic type compounds are liberated, some of which are closely related to PCDDs and PCDFs (such as the PCDPEs and PCBs) and cause interferences with the analysis for PCDDs and PCDFs unless sufficiently removed from samples. Finally, because we are dealing with both vapor and solid phase emissions, the number of operations involved in the analysis for total quantities of PCDDs and PCDFs emitted are considerable. This, coupled with the ppb or ppt concentrations with which we are dealing, makes the margin for error appreciable. For these reasons, it is imperative that we employ the most effective procedure known in each of the analytical phases - sampling, extraction, purification of the extracts, and identification and quantification.

A. Sampling

To analyze for total PCDD and PCDF emissions from a combustion source, such as a municipal incinerator, five types of samples must be obtained. These are (1) bottom ash - the ash present in the furnace after total combustion, (2) fly ash - particulates removed from the flue gas by an electrostatic precipitator (ESP), (3) smaller sized particulates which escape collection by the ESP and are present in the stack emissions, (4) resin (such as XAD-2) which adsorbs chemicals present in the gas phase of the hot stack gases, and (5) condensate, which is collected by passing the hot residual stack gas that has been filtered (to remove fine particulates) and passed through the adsorbent (to partially remove chemicals in the gas phase) through an ice-cooled trap (impinger) containing water or a water miscible organic liquid such as ethylene glycol.

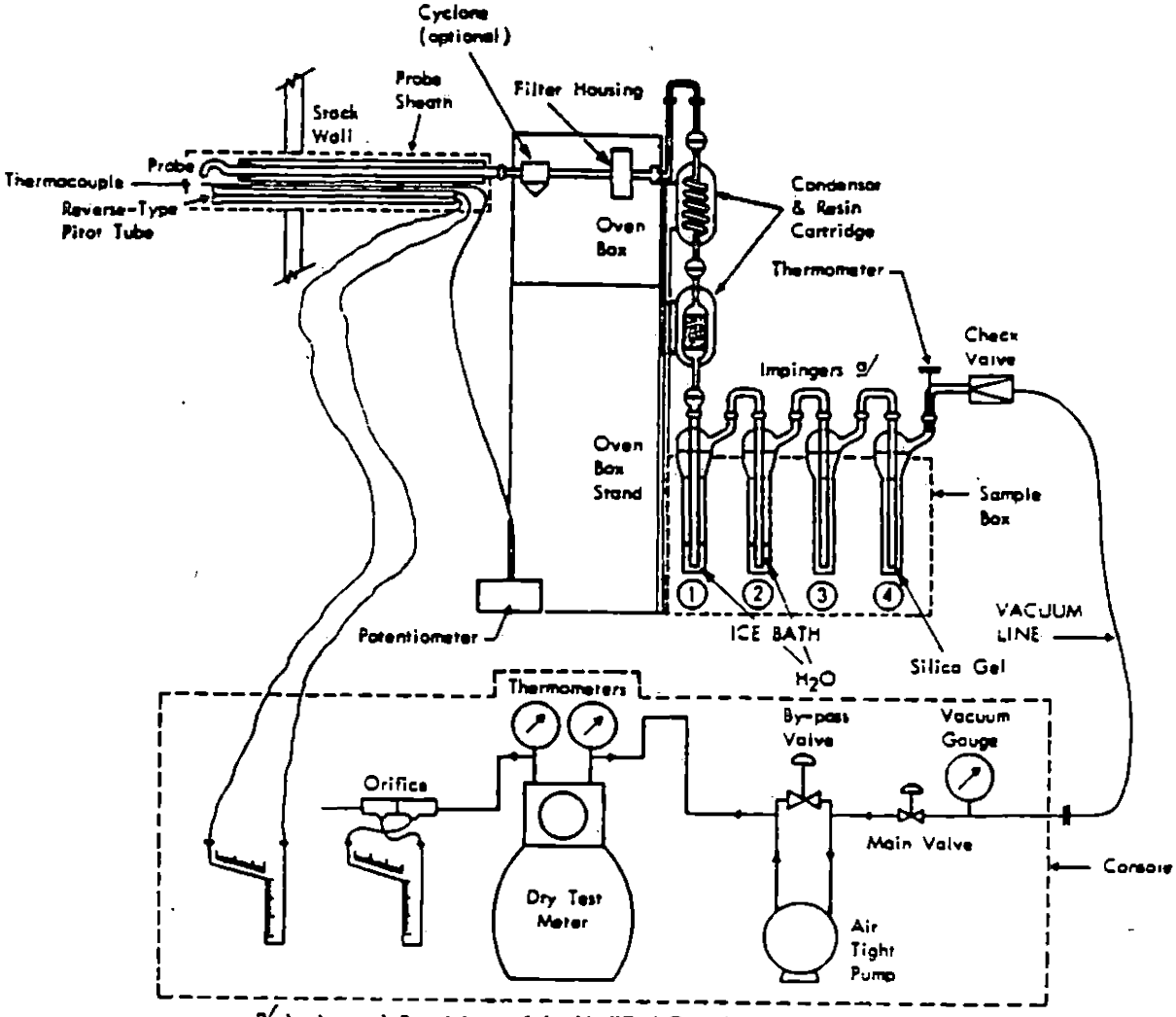
The bottom ash and fly ash samples are usually obtained with a thoroughly cleaned spoon or scoop and stored in a clean, dark-colored glass or plastic container in a cool, dark place until ready for analysis.

The method of collecting the samples associated with the flue gas has varied over the years. Currently, the most accepted collection method in the U.S. is the EPA Modified Method 5 sampling train. Figure 9 shows this sampling train.[33] The sampling probe is inserted in a sampling port in the stack wall. Stack emissions are drawn through the sampling train by means of an air pump. The stack emissions are kept hot (~120°C.) at least through a heated filter, which collects the fine particulates. The vaporous material conducted through the filter is carried by Teflon tubing to a water cooled (<20°C.) XAD-2 sorbent resin cartridge, which adsorbs gas phase compounds. The gas exiting from the resin cartridge is conducted by Teflon tubing into a bank of 4 ice-cooled

impingers. The first 2 impingers contain ~100 ml of glass distilled water for condensate trapping. The third impinger serves as a condensate overflow reservoir. The final impinger contains ~500 g. of silica gel to protect the air pump from moisture.

Much of the early work (1977 to 1980) on analysis for PCDDs and PCDFs from municipal incinerators or other combustion sources were performed by analyzing extracts of the fly ash. This was done because it was easy to obtain fly ash samples. We now know that this was not a very reliable measure of the total PCDD and PCDF emissions. Janssens and co-workers [45] indicate that only a minor fraction of the total

Figure 9
EPA Modified Method 5 Train for Organics Sampling in Combustion Processes



a/ Impingers 1, 3 and 4 are of the Modified Greenburg-Smith Type
Impinger 2 is of the Greenburg-Smith Design
Impinger 1 and 2 Contain 100 ml Water
Impinger 3 Empty
Impinger 4 Contains 200-300 Grams Silica Gel

Table 70
Measured Concentration of PCDDs in Extracts from Fly Ash[a] and Efficiencies of Extraction Methods

method[b]	TCDD	P,	HCDD	H,CDD	OCDD	Σ	REE[c]
1	110	488	1199	902	384	3083	98 } 100
1	121	457	1204	943	478	3203	102 }
2	5	11	22	19	4	61	2
2	0.3	3	19	18	4	44	1,5
3	0.2	4	5	3	7	19	0,5
3	10	90	220	330	130	780	25
4	68	251	624	417	151	1511	48
4	19	67	155	90	31	862	12
5	29	114	349	226	50	768	24
5	17	81	196	156	37	487	15
6	91	463	1089	935	279	2857	91
6	98	393	1063	966	304	2824	90
7	107	443	1145	837	355	2887	92
7	94	403	1036	836	341	2724	87

[a] Results in ppb (ng/g fly ash). [b] For description of method see text. [c] REE = relative extraction efficiency (1 = 100%).

gave the best results, with the highest extraction efficiency found on acid treatment of the fly ash prior to extraction.

Eiceman and co-workers [27] compared benzene Soxhlet extraction with benzene ultrasonic extraction of fly ash for PCDD recovery efficiency. Although ultrasonic extraction has a reduced recovery efficiency for PCDDs in comparison with Soxhlet extraction, the authors claimed their results showed that ultrasonic extraction of fly ash when combined with a rapid filtering device has acceptable precision for most routine or survey analysis for PCDDs at the ng/g (ppb) concentration levels.

Morselli, et al, [69] compared the efficiencies of the Soxhlet extraction of the PCDDs in fly ash with hexane, benzene, toluene, and xylene (mixed isomers). Table 71 shows the efficiencies of the various solvents in extracting specific PCDD isomers from fly

Table 71
Relative Efficiency of Soxhlet Extraction of PCDDs Detected in Fly Ash with Various Solvents (xylene assigned 100)

Compound	Peak No.	Extraction with Soxhlet (250 cycles)			
		hexane	benzene	toluene	xylene
Hexa-CDD 1,2,4,6,7,9	43	1.28	12.84	26.79	100
Hexa-CDD	45	3.14	16.89	39.18	100
Hexa-CDD 1,2,3,6,7,8	46	1.65	16.07	36.47	100
Hepta-CDD	52	0.95	14.19	37.40	100
Hepta-CDD	54	0.57	15.51	41.73	100
Octa-CDD	57	0.73	17.48	63.90	100

average maximum stack concentration of TCDDs is 3500 times that in the A.D. Little study, yet the estimated ground level concentration is the same.

Comparing these results with those of the Haile [33] study, the average maximum stack concentration is 8.5 times greater. If the conversion ratio from stack concentration to ground level concentration is 0.1, as used in the A.D. Little study, the degree of risk would be considerable - much greater than motorcycling. If we accept the average maximum ground level value of 0.092 pg of 2,3,7,8-TCDD/m^3, and apply the "toxic equivalents" factor of 80 for the other toxic PCDDs and PCDFs, the degree of risk is still only slightly greater than that associated with floods, tornados, and earthquakes.

Also, in 1983, Olie, et al, [77] did a risk assessment on the emissions from a municipal incinerator in Zaanstad, Holland. This is probably the best risk assessment performed to date. Using a proven dispersion modeling technique, the points of minimum and maximum concentration of emissions in the vicinity of the incinerator were determined. The air was sampled at these two points and the filters extracted and analyzed for PCDDs and PCDFs. The results are given in Table 76. In Table 77, the average concentrations of PCDDs and PCDFs in the flue gas are given together

Table 76
Air Concentrations of PCDDs and PCDFs in the Vicinity of a Municipal Incinerator (Zaanstad, Holland)

Group of isomers	location A		location B	
	conc. pg/m^3	pg tox. equ/m^3	conc. pg/m^3	pg tox. equ/m^3
TCDD	1.5	.08	.1	.005
TCDF	5	.12	.4	.009
P_5CDD	3.4	.17	.5	.025
P_5CDF	4.8	.24	.6	.030
H_6CDD	4.0	.28	2.0	.140
H_6CDF	5.2	.39	2.1	.156
H_7CDD	5.2	.26	2.1	.105
OCDD	13.3	-	.9	-
OCDF	4.8	-	.4	-
		1.54		.472

with the "toxic equivalents". Using dispersion modeling, the 119.2 ng/m^3 "toxic equivalents" in the flue gas translates to a maximum average air concentration of 38 pg/m^3 of "toxic equivalents" in the vicinity of the incinerator. Working from the daily average inhalation of a human, a maximum of 3.6 pg/day of "toxic equivalents" is inhaled. Since the acceptable daily intake (A.D.I.) is 250 pg of "toxic equivalents"/day according to Netherlands regulations, these researchers conclude that inhalation of air in

IX. SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

A. Relative Toxicity of CDDs

1. The toxicity of the various CDD isomers and homologs range all the way from the relatively non-toxic OCDD to 2,3,7,8-TCDD, which is one of the most toxic substances known as measured by animal studies. Most unusual is the fact that the toxicity of 2,3,7,8-TCDD varies greatly from one species to another. The degree of toxicity of 2,3,7,8-TCDD and the other toxic isomers and homologs to humans remains controversial.

2. Toxicological research must continue, particularly on humans. Much remains to be learned, especially regarding other potentially toxic isomers and homologs. This is very important, because 2,3,7,8-TCDD is only a minor constituent in the emissions of combustion processes; however, the "toxic equivalents" of the other PCDDs and PCDFs relative to 2,3,7,8-TCDD is 50 to 80 times greater in these emissions.

3. The structure - biological activity relationship of PCDDs is rather well elucidated; however, the ultimate target organ, whose dysfunction leads to death, is unknown. There is excellent correlation between the potency of PCDDs to induce certain enzyme systems and their toxicity. Continued histopathological research is needed.

4. The toxic PCDDs are a prototype of a large series of halogenated aromatic compounds (congeners or analogs) which includes the chlorinated dibenzofurans (CDFs), biphenyls (PCBs), biphenylenes (CBPs), azobenzenes (CABs), and azoxybenzenes (CAOBs), all of which show similar biological and toxic responses. Much more toxicity studies on these materials is needed, because some of these analogs may receive considerable environmental attention in the future.

B. Non-industrial Sources

1. The presence of significant quantities of toxic PCDDs and PCDFs in the emissions from municipal incinerators, wood-burning fireplaces and furnaces, and industrial waste incinerators has been definitely confirmed.

2. The presence of toxic PCDDs and PCDFs in fossil-fuel fired powerhouse and utility facility emissions, auto/Diesel exhausts, cigarette smoke, and charcoal grill fumes appear insignificant, but additional research is needed to definitely confirm this tentative conclusion.

3. In general, we know that PCDDs and PCDFs are not likely to be released from combustion sources if (1) the operating temperature exceeds 1200°C., (2) the resident time is sufficiently long (>1.3 sec.), (3) oxygen is in excess during combustion, and (4) material to be combusted is shredded into

E. Environmental PCDD/PCDF Input

1. With the diminished manufacture and use of 2,4,5-T and other chlorophenol derived herbicides, combustion processes, such as municipal incinerators and wood-burning fireplaces, are now suspected of being the major suppliers of PCDDs (including 2,3,7,8-TCDD) and PCDFs to the environment.

2. Although many measurements of the concentrations of PCDDs and PCDFs in emissions from the various combustion processes have been reported, only limited data necessary to estimate mass transport to the environment is available. From the limited data and considering the "toxic equivalents" of the other toxic PCDDs and PCDFs, the 52 municipal incinerators presumed operating in the U.S. may release to the environment considerably more PCDD/PCDF "toxic equivalents" per year than was released in Vietnam over a seven year period. More important, this release is over a more populated area only 2.9% the size of the area sprayed in Vietnam. Additional research is needed to provide better mass transport data from the various combustion sources to better assess potential human exposure.

F. Analytical Techniques

1. Analysis of PCDD and PCDF emissions from combustion sources is a very sophisticated operation, mainly because of the large number of organic compounds liberated at very low concentrations (ppb or ppt levels). Research must continue to search for the most effective procedures in each of the analytical phases - sampling, extraction, purification of extracts, and identification and quantification.

2. Recent studies indicate that >80% of the PCDDs and PCDFs are present in the gaseous emissions of combustion sources, not in the fly ash or bottom ash. Most of the early studies on analysis of PCDDs and PCDFs from combustion sources were performed on fly ash extracts. Earlier results obtained on the various combustion sources should be reevaluated in this light, and additional research performed on those combustion sources where confirmation of PCDD/PCDF emission levels seems warranted.

3. Protocols should be established for each analytical phase. Over the years, various methods of sampling, extraction, clean-up of extracts, and analysis have been used. Currently, the EPA Modified Method 5 sampling train appears to be the sampling method of choice, at least in this country, for stack emissions from combustion sources. For extraction of the emission samples, Soxhlet extraction with benzene or toluene seems to be the most reliable. Liquid chromatography using multilayered packed columns with various types of adsorbents is the method of choice for purification of the extracts. The level of sophistication of the clean-up procedure is dependent

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Non-Industrial Sources of Chlorinated Dibenzo-p-dioxins

I. Citation

Author(s): R. H. Stehl and L. L. Lamparski

Title: Combustion of Several 2,4,5-Trichloro-phenoxy Compounds; Formation of 2,3,7,8-Tetrachlorodibenzo -p-dioxin

Reference: Science, 197, 1008-1009, (1977)

Citation No: 99

II. Source of Dioxins

Source: Combustion of the herbicide 2,4,5-T

Sampling Techniques: Grass sprayed with 12#/acre 2,4,5-T equivalent, were taken immediately and 7 days later. Herbicide extracted with MeOH or acetone and placed uniformly on filter paper and dried. Complete combustion of paper required 5 minutes. Temperatures of 600-800°C. recorded.

Combustion flask connected to 4 gas absorber traps (all glass); 1 trap in ice bath other 3 in dry ice. Each trap is filled with glass beads. Air is drawn through system during collection.

Flow Data (mass transfer): Not discussed

Chemistry of Formation: Not discussed

Isomers Identified: 2,3,7,8-TCDD

III. Analytical Techniques Low resolution GC/MS. No further details.

Specificity: 2,3,7,8-TCDD

Sensitivity: 0.001 ug/0.5g sample of 2,4,5-T

IV. Comments (Significant Data, Graphs, etc.)

Grass and paper coated with several compounds of 2,4,5-T moiety were subjected to combustion. By using compounds that had been purified to achieve background amounts of 2,3,7,8-TCDD together with an efficient cleanup and analysis of the residue, it was possible to detect as little as 0.001 ug of 2,3,7,8-TCDD in the combustion of 0.5 gms of 2,4,5-trichlorophenoxy material.

Table 1: Combustion of material containing 2,4,5-T species.

Table 2: Combustion of grass treated with 2,4,5-T herbicide (12#/acre).

STURGEON LITIGATION

FROM THE FILES OF Dr. Harold Bibb

THIS FILE HAS BEEN REVIEWED BY Katherine Hill

FOR:

- Documents describing the presence or potential presence of dioxin in Monsanto products.
- Documents referring to the toxicity of any Isomer of dioxins.
- Documents referring to the toxicity of any Chlorophenol or Chlorophenol product.
- Documents referring to health effects as they relate to the above.

DATE April 22, 1985

Weinberg Consulting Group Inc.
2828 Pennsylvania Avenue NW, Suite 301
Washington, D.C. 20007
(202) 342-8077

MAY 2 [illegible] 84

ATTORNEY'S WORK PRODUCT
PRIVILEGED AND CONFIDENTIAL

May 25, 1984

Roger Tompkins, Esquire
Bowles, McDavid, Graff & Love
Commerce Square, Suite 1600
Charleston, WV 25301-1386

Dear Roger:

When we visited you in Charleston, we briefly discussed the validity of Alistair Hay's cell transformation study on dioxin. You noted the need for an audit of this study, and that is the subject of this letter.

We have found, by examining writings and depositions, a number of basic problems with Dr. Hay's study. His purported expertise in toxicology (mutagenicity, carcinogenicity) is based on this single experiment; a short term bioassay for carcinogenicity. In actuality, Dr. Hay's only role was to suggest the test and provide the sample material for analysis. He does not understand the experimental protocol or interpretation. For example, Dr. Hay claims that the results show TCDD to be a mutagen, when actually, this test indicates potential carcinogens.

How prove this? good

The cells used in this assay were premalignant (already initiated so only second stage carcinogenesis could be observed). In his test, phenotypic (non-genetic) response was ascertained by growth in soft agar. To show permanent, genetic response, cell colonies should have to be injected into animals and observed for tumor growth.

Monsanto

Monsanto Company
800 N. Lindbergh Boulevard
St. Louis, Missouri 63167
Phone: (314) 694-1000

June 19, 1984

Weinberg Consulting Group Inc.
2828 Pennsylvania Avenue, N.W.
Suite 301
Washington, D.C. 20007

Dear Myron:

I've attached some literature which Bo Holmstedt sent Dave Snively last week. We've seen most of the articles he's sent, but haven't seen the monograph by Tuula Thunberg which ties it all together. The more I've gotten into it the more obsorbing its become.

The central thesis is that most, perhaps all of the symptoms associated with 2,3,7,8-TCDD toxicity are also induced by hypovitaminosis A. According to Thunberg the symptoms in common between vitamin A deficiency and 2,3,7,8-TCDD toxicity are:

1) Delayed symptoms
2) Failure of normal growth and general kachexia
3) Keratosis
4) Acne
5) Epithelial lesions and infections
6) Immunosuppression
7) Reproductive abnormalities
8) Teratogenesis
9) Skeletal lesions
10) Lesions in the nervous system or neurological symptoms

I'm sure that you are aware that only a small fraction of the bodies vitamin A is used in the visual cycle with the majority performing a multitude of body functions many of which are poorly understood and probably many more unknown. It's interesting that it is considered a fairly potent anticarcinogen which suggest that its absence would have a promotional effect as seen with 2,3,7,8-TCDD.

The importance of these observations is that Thunberg and others have looked at vitamin A storage in rats dosed with 2,3,7,8-TCDD (as well as with other enzyme inducers). They find that a storage in rat liver is considerably reduced by 2,3,7,8-TCDD.

Thunberg also indicates that the amount or extent of reduction in various animal species correlates with 2,3,7,8-TCDD toxicity in the various species ie for a given 2,3,7,8-TCDD dose (at the right level) a more severe reduction in vitamin A storage is seen in male Hartley guinea pigs than in Syrian hampsters.

This would seem to suggest that not only is "dioxin" toxicity lacking in uniqueness, but that the biochemical effect is one seen to some extent in this country but a chronic problem in many places in the third world; that of hypovitaminosis A

Let me know your thoughts.

Sincerely,

Allan M. Ford, Ph.D.

P.S. If I haven't discussed this with Dr. Karch by the time you get this, I will shortly.

cc: Nathan Karch
W. McCarville
G. Roush
J. Wilson
K. Johnson

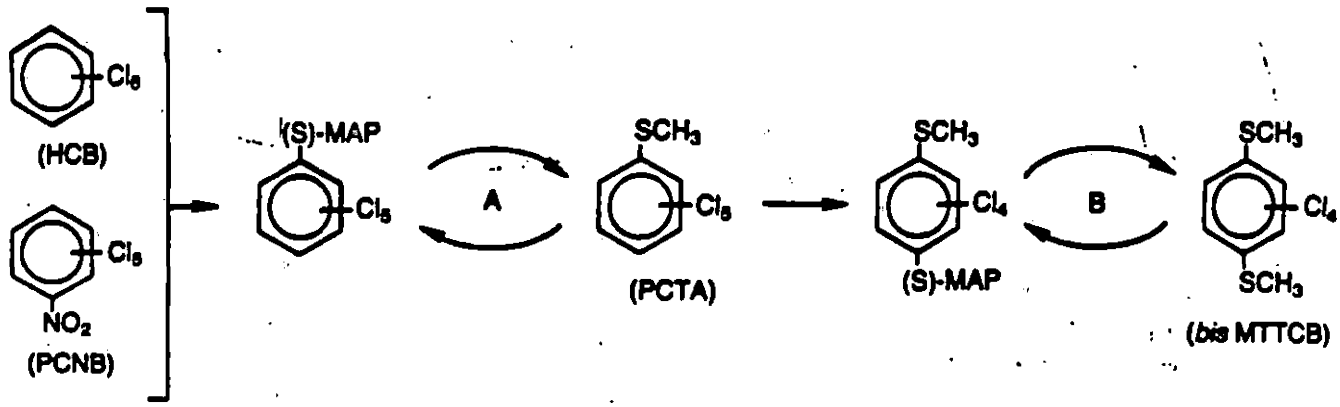

Fig. 2. Methylthiolation of hexachlorobenzene (HCB), pentachloronitrobenzene (PCNB), and pentachlorothioanisole (PCTA); also the turnover of CH_3S-groups in PCTA and bismethylthiotetrachlorobenzene (bisMTTCB). Pathways A and B, indicated by the curved arrows do not represent an equilibrium. The forward arrows represent a C–S lyase-mediated methylthiolation. The reverse arrows represent either enterohepatic circulation or environmental circulation of the methylthio-containing metabolites (pathway C, Fig. 1).

able as substrates for the C–S lyases. This may be possible in the case of the microfloral C–S lyases by analysis of the divalent sulfur present in the bile of animals on various diets. The divalent sulfur of interest would be present in MAP metabolites and *S*-glucuronides.

Nonpolar methylthiolation products

A further consequence of the existence of the methylthiolation pathway is the generation of nonpolar compounds (CH_3S-containing) from the polar conjugates that were formed to facilitate excretion of the original xenobiotic from the cell. These nonpolar methylthiolation products behave as new xenobiotics in the biosphere. Examples are the CH_3S-, CH_3SO- and CH_3SO_2-containing metabolites produced in the metabolism of polychlorinated biphenyls[15], DDT[15], hexachlorobenzene (HCB), and pentachloronitrobenzene (PCNB).

Recent reports concerning the metabolism of HCB, PCNB, and pentachlorothioanisole (PCTA) indicate that methylthiolation may function to form CH_3S-containing residues that persist and turnover in the environment. This process is outlined in Fig. 2.

HCB and PCNB are assumed to be metabolized in part to PCTA via the methylthiolation pathway. PCTA is, in turn, known to be metabolized to bis-methylthiotetrachlorobenzene (*bis*MTTCB) by microflora-mediated methylthiolation[16]. *Bis*MTTCB is metabolized through the same process. However in this case, a CH_3S-group, and not a chlorine, is replaced[17] which indicates that the CH_3S-group is a better leaving group for GSH conjugation than the remaining chlorine groups. This same process, i.e. turnover of CH_3S-groups, functions in the metabolism of PCTA and other methylthio-containing compounds. Turnover of CH_3S-groups by the outlined process (Fig. 2 and sequence C, Fig. 1) requires the utilization of GSH and results in the transient formation of thiols that again utilize SAM.

Turnover of methylthio-groups in *bis*MTTCB metabolism has been demonstrated only in rats[17]. However, there is evidence that the processes shown in Fig. 2 can occur, at least in part, not only in mammals, but also in fish, protozoa, fungi, and plants. The ability of a number of species to carry out any one or all of the steps shown in Fig. 2 indicates that various species could be involved in the formation and turnover of *bis*MTTCB in the environment. Therefore, methylthiolation of HCB, PCNB, and PCTA may be responsible for the high residues of *bis*MTTCB and PCTA present in mussels collected along the coast of Holland. Such residues represent another potential source of thiols that could be involved in the toxic processes considered earlier.

Methylthiolation does function to increase the turnover of residues from HCB in rats. About 70% of the ^{14}C from oral doses of [^{14}C]HCB (5 mg kg^{-1}) were left in the bodies of rats seven days after dosing[18]. Three days after oral doses (15 mg kg^{-1}) of either [^{14}]PCTA[16] or [^{14}C]*bis*MTTCB[17], only 5% of the ^{14}C remained in the bodies. Therefore, methylthiolation can function to increase the turnover of HCB-derived residues in rats. However, the rate of turnover and ultimate fate of these residues in the environment is not known. In another example, however, methylthiolation of polychlorinated biphenyls produces CH_3SO_2-containing metabolites that persist in lung tissue[11].

Extension of this pathway to the metabolism of methylthiolation products from other xenobiotics indicates that CH_3S-, CH_3SO- and CH_3SO_2-containing compounds can be persistent or terminal residues in the environment. The formation of such products could be a rate-limiting step in the ultimate biodegradation of many xenobiotics. The wide distribution of enzymes involved in methylthiolation and the number of species capable of being involved in methylthiolation indicate that methylthiolation products are present in the food chain.

Conclusions

In this review we have described the existence of a novel pathway for the generation of methylthiolated metabolites of xenobiotics involved in C–S lyase activity in the intestinal microflora. Mercapturic acid pathway metabolites of xenobiotics have been shown to produce metabolites of toxicological significance during the process of methylthiolation and this process may therefore be regarded as a pathway which involves metabolic activation of xenobiotics by the intestinal microflora. Other examples of the role of the gut bacteria in generation of toxic metabolites are cleavage of cycasin, reduction of nitroaromatics such as nitrotoluene, as well as formation of certain mutagenic compounds from unknown precursors. The significant metabolism of xenobiotics by the intestinal microflora is not surprising in view of the quantitatively important breakdown of endogenous compounds such as steroid hormones which has been shown to be effected by the gut bacteria[19,20]. Conceivably, the metabolic

mic myocardium. The mechanism by which antianginal drugs attenuate the coronary occlusion-induced pH decrease, however, remains to be investigated.

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Kazuo Ichihara, Ph.D. is an Associate Professor of Pharmacology, Asahikawa Medical College. After graduating from the Faculty of Pharmaceutics, Hokkaido University in 1973, he worked on the myocardial metabolism in the ischemic heart with Dr Abiko at the Fukushima Medical College for one year and at the Asahikawa Medical College from 1974 to the present. He did postdoctoral research training in the Department of Physiology, Hershey Medical Center of the Pennsylvania State University (1979–1981).

Kenji Sakai, Ph.D. is an Instructor of Pharmacology, Asahikawa Medical College. After graduating from the Tohoku College of Pharmacy (1978), he joined Dr Abiko in Asahikawa.

Mercapturic acid pathway metabolites of xenobiotics:

generation of potentially toxic metabolites during enterohepatic circulation

Jerome Bakke and Jan-Åke Gustafsson*

*Metabolism and Radiation Research Laboratory, ARS, US Department of Agriculture, State University Station, Fargo, ND 58105 USA. *Department of Medical Nutrition, Karolinska Institute, Huddinge University Hospital, S-141 86 Huddinge, Sweden.*

The body detoxifies many xenobiotics via the mercapturic acid pathway (MAP). Unfortunately, in some cases this process provides intermediate metabolites that are substrates for mutagen/toxin-forming reactions – in particular reactive thiols. Jerome Bakke and Jan-Åke Gustafsson describe this process of methylthiolation and the novel metabolic pathway involved. They highlight the increasing load of xenobiotics imposed by industrial processes and the toxicological consequences of their methylthiolated metabolites in individual organisms and the biosphere in general.

Many xenobiotics, including known carcinogens, are metabolized in the mercapturic acid pathway (MAP, defined in Fig. 1) after conjugation with glutathione (GSH). This conjugation is assumed to effect the detoxification of the xenobiotic upon excretion of the GSH-conjugate from the cell in which it was formed[1]. However, catabolites of some MAP metabolites have been shown to be mutagens[2,3], to be toxic to the kidney[3], and to bind to macromolecules, i.e. bound residues[4]. The toxicological activities of this catabolic system are thought to reside in formation of reactive thiols. These thiols are formed upon cleavage of the cysteine conjugates by cysteine conjugate 7-lyase (C–S lyase[5]). This catabolic system is outlined in Fig. 1 (follow pathway A).

Methylation of the thiols to form methylthio-(CH_3S-)containing metabolites is another detoxication step. However, methylation is only a transient detoxication because the resultant CH_3S-, CH_3SO- and CH_3SO_2-containing metabolites exhibit polarities similar to the parent xenobiotic; these can therefore be absorbed into cells where they must once again undergo detoxication (e.g. pathways C, Fig. 1). In essence, the methylation creates new xenobiotics that can add not only to the detoxication burden of the organisms that formed them but also to that of subsequent compartments of the environment. For convenience, the overall process outlined in Fig. 1 will be termed methylthiolation.

Two metabolic processes are known which result in the methylthiolation of xenobiotics. These are the C–S lyase pathway described above (pathway A, Fig. 1), and the reaction of activated intermediates of xenobiotics with methionine and/or methionyl residues of proteins to form sulfonium ions (sequence B, Fig. 1). These sulfonium ions must then dissociate to form

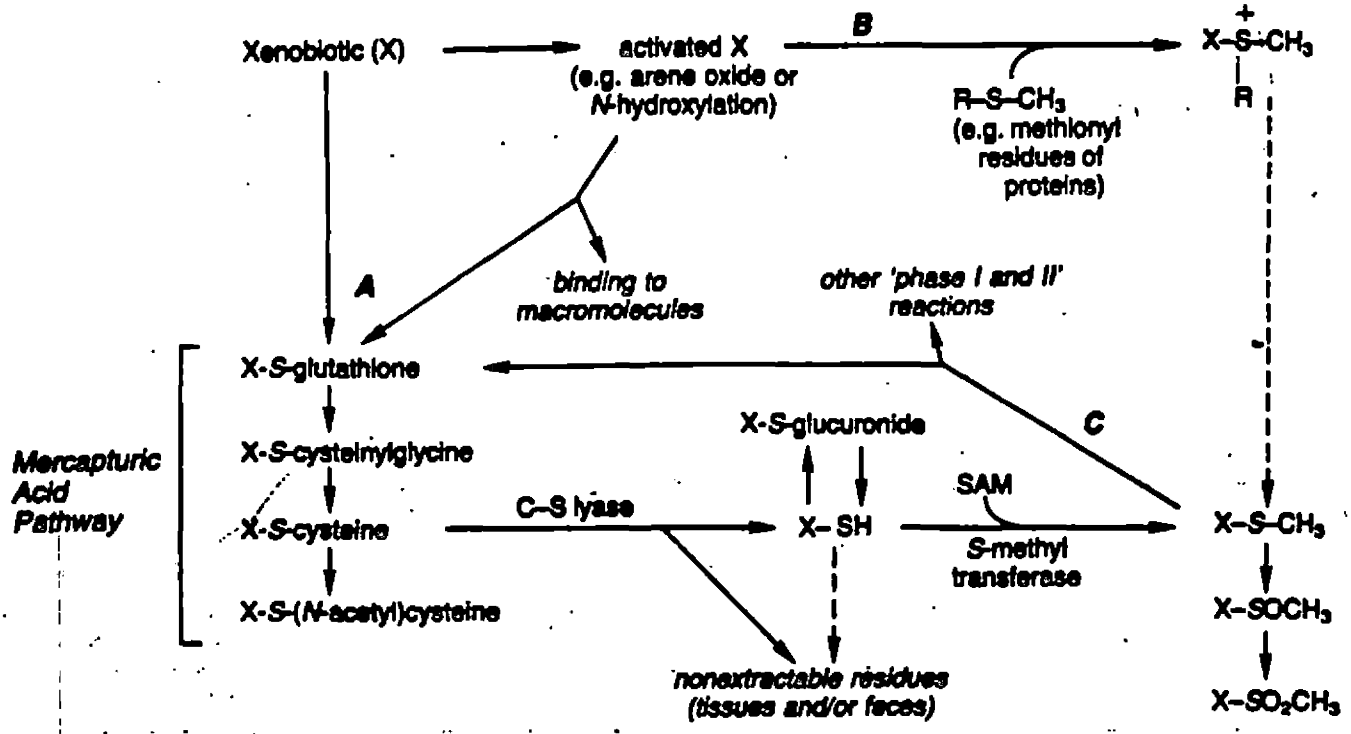

Fig. 1. Pathways leading to the introduction of methylthio-groups into xenobiotics via the mercapturic pathway (A) and via sulfonium ion formation (B). Pathway A appears to dominate quantitatively. Pathway C represents not only enterohepatic circulation, but also environmental circulation and fate of methylthio-containing metabolites. Dashed arrows designate suspected but unproven pathways. SAM = S-adenosylmethionine.

CH_3S-containing metabolites.

Sulfonium ion pathway

The sulfonium ion pathway has not yet been shown to produce, *in vivo*, CH_3S-containing metabolites that are excreted in either urine or feces. However, methyl sulfides of 2-acetylaminofluorene were isolated after alkaline hydrolysis of proteins isolated from livers of rats that were dosed with 2-acetylaminofluorene[6]. The quantitative importance of the sulfonium ion pathway with respect to production of methylthio-containing compounds from the above xenobiotics or other activated xenobiotics has not been determined, but it is considered to be less prevalent than the C–S lyase pathway. The sulfonium ion pathway is of toxicological importance because it results in binding of xenobiotics to tissue macromolecules.

The prevalence of methylthiolation in xenobiotic metabolism is demonstrated by the varied structures that are known to be metabolized to CH_3S-containing metabolites (Table I). Most of the xenobiotics that are metabolized to CH_3S-containing metabolites are also metabolized in the MAP (noted in Table I).

C–S lyase pathway

The C–S lyase-mediated methylthiolation pathway (sequence A, Fig. 1) is known to function in plants and mammals, and probably functions in fish, protozoa and fungi. The key reaction in this pathway is catalysed by C–S lyases which cleave *S*-substituted cysteine conjugates to yield pyruvate, ammonia and the thiols of the original xenobiotics. *In vivo*, the thiols are subsequently methylated or conjugated with glucuronic acid. In the latter case, the *S*-glucuronides can undergo enterohepatic circulation during which the thiols are regenerated by intestinal glucuronidases. The thiol-containing aglycones can be absorbed, methylated and excreted. In many cases, the CH_3S-containing metabolites are oxidized to the methylsulfinyl (CH_3SO-) and methylsulfonyl (CH_3SO_2-) analogues.

In mammals and possibly other bile-producing species, two chemically identical, but physiologically different, C–S lyase-mediated methylthiolation pathways operate. This also applies to species which exist in symbiosis with an intestinal microflora. One pathway is mediated by tissue C–S lyases[7,8] which can cleave *S*-cysteine conjugates formed on the first pass of xenobiotics through the tissues. The other pathway is mediated by C–S lyases located in the intestinal microflora[5,9]. These latter C–S lyases can function on both dietary and biliary MAP metabolites.

Specificity of C–S lyases

The tissue and microfloral C–S lyases exhibit a specificity for *S*-cysteine conjugates[7]. In addition, the tissue C–S lyases, *in vitro*, exhibit specificity for *S*-cysteine conjugates with unsaturation or aromaticity at the carbon of the xenobiotic moiety that is bonded to the cysteinyl sulfur[8]. This specificity has not been observed with microfloral C–S lyases; however, it is not known how many different C–S lyases are present in the intestinal microflora. The differing specificities of the tissue and microfloral C–S lyases are important because they determine, in part, the quantitative roles played by the tissue and microfloral pathways. Because of the broader specificity of the microfloral C–S lyases and the presence of the metabolic activities of the microflora discussed below, any MAP metabolite excreted with the bile or present in the diet is a potential substrate for microfloral C–S lyases.

Of the two C–S lyase mediated pathways, we believe the pathway mediated by the microflora to be quantitatively the most important in view of the levels of MAP metabolites of various xenobiotics in the bile and the ability of intestinal systems to produce *S*-cysteine conjugates from other MAP metabolites. For example, all the MAP metabolites of propachlor (2-chloro-*N*-isopropylacetanilide, a herbicide), including the sulfoxide of the mercapturic acid, produced varying amounts of the thiol (2-thiolo-*N*-isopropylacetanilide) upon their incubation with the intestinal microflora from pigs[5]. From these results, it was concluded that peptidases, acylases and

reductases present in the intestine were functioning to form the *S*-cysteine conjugate from the MAP metabolites. Similar tissue systems would not supply these levels of *S*-cysteine conjugates to the tissue C–S lyases because of excretion of the other MAP metabolites with bile and urine before metabolism back to the cysteine conjugate.

Toxicological implications

The biological function of the C–S lyase is not known. However, toxicological consequences have been attributed to their action. These consequences include anemias[10], renal tubular adenocarcinomas[3] and nephrotoxicity caused by C–S lyase cleavage of halogenated vinyl-*S*-cysteine conjugates to reactive thiols. These thiols have been shown to bind to proteins and inactivate the enzyme. C–S lyase cleavage of MAP metabolites has also been proposed to produce nonextractable fecal[4,5] and plant residues resulting from metabolism of pesticides. The type of chemical bonding present in these residues has not been determined. Methylthiolation of chlorinated hydrocarbons has also been shown to result in methylsulfonyl-containing residues in animal tissues[11]. Here again, the significance of these residues is unknown other than that some drug metabolizing enzymes are induced by the administration of CH_3S-containing metabolites of *m*-, and *p*-chlorobenzene.

Another possibility for an adverse role of C–S lyases is the hemolytic anemia produced in ruminants fed a diet containing high levels of kale[10]. The toxic factor is *S*-methylcystein sulfoxide (present in high concentrations in brassicas) which could be reduced by the microflora to *S*-methylcysteine and then serve as a substrate for C–S lyases to produce high levels of methanethiol.

The products from both C–S lyase cleavage and methylthiolation therefore exhibit properties generally associated with toxic compounds, i.e. binding to tissue macromolecules and the formation of persistent residues. Carcinogens are believed to exert their toxic effect in binding of reactive metabolites to macromolecules, and many carcinogens are metabolized via arene oxides to MAP metabolites. It is therefore possible that the products and/or intermediates of the methylthiolation pathways could be involved in carcinogenesis.

Further metabolism

The magnitude of the burden of xenobiotics available to the C–S lyases as cysteine conjugates is now known. However, considering the varied structures that are conjugated with glutathione (GSH), the prevalence of biliary secretion of the resultant MAP metabolites, and the activity of the intestinal microflora, we can assume that there is a high level of production of thiols in the intestinal lumen. These thiols, because of their reactivity, must be disposed of by further metabolism. There appear to be three general routes of disposal: methylation and excretion with the feces; methylation or glucuronidation and excretion with the urine; and incorporation into the feces as nonextractable residues. Two of these routes involve *S*-methylation which is presumed to be catalysed by *S*-methyltransferases using *S*-adenosylmethionine (SAM) as the methyl donor. The physiological locations of the *S*-methyltransferases involved in methylthiolation have not been determined. *S*-methyltransferases are present in most tissues including the intestinal mucosa[12]. However, they have not been shown to be present in intestinal contents[5,12]. This indicates that the intestinal mucosa would be the first tissue in which thiols formed in the intestinal lumen could be methylated. The consumption of methyl donors (SAM) by thiols produced in the intestine, together with the production of thiols capable of binding to tissue macromolecules, could work individually, together, or in concert with other carcinogens produced or present in the intestine to produce the toxic effect described below.

Carcinogenesis

According to Farber[13], three conditions are conducive to the occurrence of chemical carinogenesis, i.e. proliferating cells, initiation of the cancer, and promotion of cancerous growth. These three conditions could exist in the intestine under the stress of a continuous supply of a variety of cysteine conjugates to the intestinal microflora. Cell proliferation is a natural process in the intestinal mucosa because the mucosa is constantly replacing cells sluffed off into the intestinal lumen. Initiation of carcinogenesis could be effected by the binding of reactive thiols produced by C–S lyase cleavage of appropriate cysteine conjugates excreted with the bile or present in the diet. Promotion of the tumor could be mediated by the production of a constant supply of various thiols either by the action of the thiols themselves or, in view of the fact that diets deficient in methyl donors such as choline and methionine promote carcinogenesis[13], by producing a localized deficiency of SAM required for their methylation.

In this model, initiation could also be effected by compounds formed as a result of the action of the microflora on other biliary metabolites, such as the regeneration of the parent xenobiotic as Renwick and Drasar[14] have shown for benzo(a)pyrene. Upon incubation of biliary metabolites of benzo(a)pyrene (which would presumably contain MAP metabolites) with intestinal microflora, they found benzo(a)pyrene was formed. Promotion of carcinogenesis need not be attributed to any specific MAP metabolite, but to the burden of thiol precursors excreted with the bile or present in the diet. This description of chemical carcinogenesis could be extended to other tissues, such as liver and kidney, where C–S lyases are present. Also, thiols produced by the flora which bypass the mucosa *S*-methyltransferase could be involved in promotion of carcinogenesis in other tissues. To test this model requires knowledge of both the types and quantities of thiol precursors that are avail-

TABLE I. Xenobiotics shown to be metabolized to methylthio-containing metabolites.

Polychlorinated biphenyls	Caffeine
DDT	2,6-Dichlorobenzonitrile
Biphenyl	2,6-Dichlorobenzamide
Naphthalene	2,6-Dichlorothiobenzamide
Phenanthrene	2-Chloro-6-methylthiobenzonitrile
Carbamazepine	1,1,1-Trifluoro-*N*-[2-methyl-4-(phenylsulfonyl)phenyl]methanesulfonamide
Bromobenzene	Acetylaminofluorene
Phenacetin	*O,O*Diethyl-*O*-(3,5,5-trichloro-2-pyridyl)-phosphorothioate
Acetaminophen	2-Acetamido-4-chloromethylthiazole
Hexabromobenzene	3-(5-Nitro-2-furyl)-2-(2-furyl)acrylamide
Hexachlorobenzene	Polychlorinated dibenzo-*p*-dioxins
Pentachloronitrobenzene	3-Hydroxyxanthine
Pentachlorothioanisole	Propranolol
Bromazepam	1,1-Dichloroethylene
1-Allyl-3,5-diethyl-6-chlorouracil	
2-Chloro-*N*-isopropylacetanilide	

[a] Methylthio-containing metabolites isolated after a hydrolytic step.

processes occurring in the intestinal microflora may be influenced by dietary habits and, consequently, the findings presented in this article may be of relevance for the important issue of nutrition and cancer. It is possible that further investigations concerning the influence of dietary components, e.g. fiber, on the metabolism of xenobiotics, and perhaps also of endogenous compounds, may help to define a diet that could minimize the formation of toxic metabolites during enterohepatic circulation.

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New trends in lower urinary tract pharmacology

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The very common disorder of urinary incontinence has proved resistant to satisfactory pharmacological therapy. Karl-Erik Andersson describes how recent developments in understanding of the complex neuromuscular mechanisms which control urinary function has opened up new areas for pharmacological investigations.

In the last decade, disturbances of the function of the lower urinary tract have attracted an increasing interest, reflected by, e.g., the contributions to the annual meetings of the International Continence Society. These disturbances can manifest themselves as urinary incontinence. This is a common condition; in Britain it was estimated that over two million people suffer from the disorder[1]. The efficacy of available methods of treatment is often unsatisfactory. Only a few women with urine leakage precipitated by stress (e.g. heavy lifting, coughing) are suitable for surgical treatment, and pharmacological treatment is restricted to a few drugs with limited efficacy. Another form of incontinence, motor urge incontinence, is characterized by involuntary bladder emptying associated with uncontrolled contractions of the bladder ('unstable bladder'). Many drugs have been tried for inhibition of the bladder contractions in this condition, but their efficacy is limited and adverse reactions common[2].

The lack of satisfactory drug treatment in urinary incontinence depends ultimately on lack of knowledge about the basic physiological mechanisms regulating the function of the bladder and urethra, both under normal conditions and in different types of disease processes and injuries. It has therefore been one of the aims of research in this field to increase the amount of information concerning the innervation and receptor functions in the bladder and urethra to secure a basis for development of rational pharmacological and surgical methods of treatment. It is generally accepted that vesicourethral function in micturition is controlled by an extremely complex neuromuscular mechanism. However, in the last few years much new information has been compiled describing the detailed anatomy and innervation of this region[3–5], opening new ways for further physiological and pharmacological investigations.

Stress incontinence and urethral α-adrenoceptors

At least three factors are of importance for maintenance of continence in women; the urethral resting pressure, the intra-vesical pressure, and the transmission of the intra-abdominal pressure to the proximal part of the urethra[6]. In stress incontinence there is probably a defect in the transmission of the intra-abdominal pressure to the urethra. Partly because of this, the urethral resistance to flow is not enough to prevent leakage of urine when the intra-abdominal pressure is rapidly increased. There is no abnormal activity in the bladder. As women with stress incontinence have a lower than normal intra-urethral pressure, the aim of pharmacological treatment has been to increase this pressure. Several investigators have stressed the importance of the sympathetic nervous system to maintain intra-urethral pressure – it is effectively lowered by α-adrenoceptor blockers, such as phenoxybenzamine, phentolamine and prazosin, and increased by α-adrenoceptor agonists. Clinically, orally-effec drugs such as phenylpropanolamine and ephedrine are the most widely used[2]. These drugs have no urethral