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ABSTRACT

Polychlorinated dibenzodioxins and dibenzofurans ("dioxins") include compounds that are environmentally objectionable because of their toxicity, lipophilicity, and persistence. Consequently, their discovery in effluents, sludges, and pulps led to a massive research effort by the pulp and paper industry. This research, which required sophisticated analytical methodology, has been successful. It has resulted in the identification of the chlorination stage as the source of the dioxins, their unchlorinated analogs as important precursors, and direct substitution of chlorine as an important mechanism of formation. A variety of control strategies have been developed. Some of these decrease the amount of precursors entering the chlorination stage, while others decrease the amount of chlorine used. Their implementation has resulted in a reduction of more than 90% in the amount of dioxins released by North American bleached pulp mills. This paper reviews the subject of dioxins as it relates to the paper industry, with reference to their nature, analysis, toxicity, risk assessment, mechanism of formation, control strategies, and current discharge levels.

INTRODUCTION

Perhaps no other event has led to so much public attention being focused on the paper industry as the finding, in 1985, that fish collected downstream from paper mills contained detectable levels of dioxins.[1] The finding was unexpected, since studies at some of the same locations only two years earlier had failed to show the presence of dioxins at levels which exceeded the then current detection limits in the parts per billion range. Advances in analytical methodology enabled the 1985 discovery at levels in the parts per quadrillion to parts per trillion range.

The extreme smallness of the concentrations detected went unnoticed by environmentalists and the general public. The alarming fact was that substances viewed as being among the most toxic materials on earth were being unnecessarily released to the environment. The stage was clearly set for action to solve the problem.

What followed was a concerted voluntary effort by the pulp and paper industry to identify and remove the source of the emissions. This effort, which involved the expenditure of enormous resources, in terms of both money and manpower, was largely successful. Nonetheless, the course of the industry's technological development has been permanently altered.

A corroborative study of discharges from five representative mills, conducted in 1986 and 1987, confirmed that dioxins were produced during the pulp bleaching process.[2] This was followed by a cooperative EPA/industry project to analyze the effluents, sludges, and pulps at all 104 U.S. bleached pulp mills.[3] Key components of subsequent industry efforts have included mill studies by the National Council of the Paper Industry for Air and Stream Improvement (NCASI) and the Institute of Paper Science and Technology.

The objective of this paper is to provide an overview of these and other studies of dioxins in the paper industry, together with a review of relevant information on their properties, toxicity, analysis, occurrence, and current discharge levels. Emphasis will be on investigations of the source of the discharges and strategies for their control.

WHAT ARE DIOXINS?

The term "dioxins" is commonly used to collectively denote derivatives of dibenzo-p-dioxin (DBD) and dibenzofuran (DBF) obtained by substituting chlorine atoms for the hydrogen atoms of the parent molecules. Because DBD and DBF molecules each contain eight hydrogen atoms, not all of which are equivalent, there are many ways in which any given number of them can be substituted by chlorine. The result is that there are 75 different possible chlorinated dibenzo-p-dioxins (CDDs) and 135 different chlorinated dibenzofurans (CDFs). The commonly encountered members of these families of compounds (congeners) contain four or more chlorine atoms and are referred to as polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). **Figure 1** shows the molecular structures of DBD, DBF, 2378-TCDD, and 2378-TCDF.

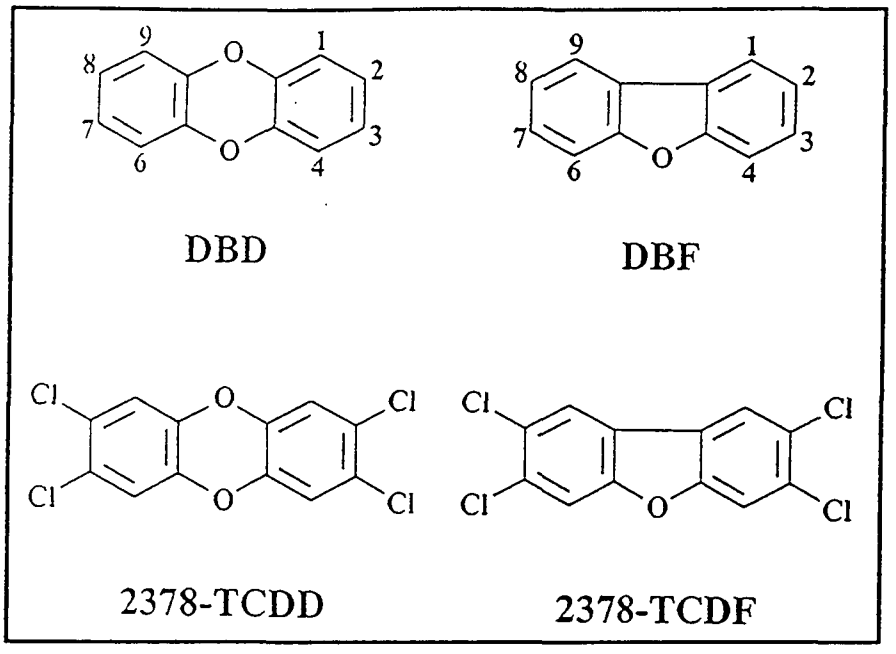

Figure 1. Molecular Structures of Dibenzo-p-dioxin, Dibenzofuran and Their 2378 Tetra-chlorinated Derivatives.

Certain PCDDs and PCDFs are highly toxic to laboratory animals, but there is much variation between congeners in this regard. In fact, only 7 of the 75 CDDs and 10 of the 135 CDFs are considered toxic and, among these, potency varies by about three orders of magnitude. To enable comparison of the toxicities of different mixtures of PCDDs and PCDFs, a scale of relative toxicities has been defined.[4] Numbers on this scale, called toxicity equivalency factors (TEFs), have been assigned to the 17 congeners of interest. The relative toxicity of a mixture can then be estimated by multiplying the concentration of each congener by its TEF and summing the products. Since 2378-TCDD is assigned a TEF of 1.0, the resulting sum is the concentration of 2378-TCDD which would have the same toxicity as the mixture in question. It is expressed as 2378-TCDD toxicity equivalents (TEQ). The TEFs are shown in **Table I**.

In the present paper, the singular term "dioxin" will be used to denote 2378-TCDD or the sum of a collection of CDD and CDF congeners expressed as TEQ.

PCDDs and PCDFs have extremely low water solubility (a few nanograms per liter [parts per trillion, ppt]) and vapor pressure values; they are highly lipophilic, having an octanol/water partition coefficient of greater than one million; and they are strongly sorbed by solids such as soils and organic matter. They are very stable chemically,

being resistant to oxidation, hydrolysis and biodegradation. These properties, together with their toxicity, make them highly objectionable from the environmental standpoint, since they result in persistence and bioaccumulation.

Table I. International Toxicity Equivalency Factors[4]

Congener	PCDD	Isomers	PCDF	Isomers
2378-Tetra-	1.0	1	0.10	1
2378-Penta- (12378)	0.50	1	0.05	1
2378-Penta- (23478)	----	-	0.50	1
2378-Hexa-	0.10	3	0.10	4
2378-Hepta-	0.01	1	0.01	2
Octa-	0.001	1	0.001	1

ANALYSIS

Because dioxins are usually present at extremely low concentrations, their quantitative determination presents a difficult challenge. The task is usually further complicated by the presence of complex mixtures of interfering substances in the samples to be analyzed. Consequently, accurate dioxins analysis requires a combination of effective methods for concentrating the sample, enriching it in the analytes of interest by selectively removing interfering materials, separating the analytes of interest from remaining interfering materials and from each other, detecting the analytes with high sensitivity and specificity, and quantitating them. Methods incorporating all of these elements have evolved over the past 30 years to the present state, characterized by detection limits in the low parts per quadrillion (10^{-12} g/L) range.[5]

Nearly all of these methods employ solvent extraction, multistep analyte enrichment ("cleanup") protocols based on column chromatography, high resolution gas chromatographic separation detection by mass spectrometry (MS), and isotope

dilution for quantification and quality assurance. Many laboratories use high resolution MS to achieve low detection limits and high isomer specificity, but low resolution MS can be employed after efficient cleanup. The range of methods in use has been reviewed by Firestone.[6]

Two methods that have been used extensively for pulp and paper industry samples are the National Council of the Paper Industry for Air and Stream Improvement (NCASI) method[7] and the United States Environmental Protection Agency (USEPA) Method 1613A.[8] These have recently been compared with a newer method developed by the United States Food and Drug Administration (USFDA).[9] They serve to illustrate the principal features of most of the methods currently in use.

According to the NCASI method, the pulp or paper sample is ground in a Wiley mill and extracted with 95% ethanol in a soxhlet extractor. Prior to extraction, ^{13}C-2378-TCDD (2.0 ng) and ^{13}C-2378-TCDF (2.0 ng) are added as internal standards. After extraction is complete, ^{37}C-2378-TCDD (1.0 ng) is added to monitor the efficiency of the cleanup process. This consists of silica gel filtration followed by passage successively through columns of the following: NaOH-silica, silica, H_2SO_4-silica, acid alumina and activated carbon. After concentration to 20 µL, and before injection into the GC/MS system, ^{13}C-1234-TCDD (2.0 ng) is added as recovery standard to quantify recovery of the internal standards. The EPA method differs principally in regard to the extraction method and the solvent used for extraction (toluene).

REGULATION, RISK ASSESSMENT, AND TOXICITY

Regulating agencies are charged with protecting the public from unnecessary risks that may otherwise be incurred as a result of industrial activity. In a given situation, they do this by estimating both the toxicity of a material and the public's exposure to it. Exposure is the product of concentration and intake, and risk is the product of exposure and potency (toxicity). Consequently, at least in principle, risk can be regulated by regulating the discharge levels that determine concentration.

This simple regulatory model assumes that toxicity can be unambiguously evaluated. In reality, this is seldom true. This is nowhere better exemplified than in the case of dioxins. These compounds are singularly toxic toward laboratory animals, resulting in high levels of cancer incidence, but ample evidence exists for the supposition that toxicity toward humans is much less than the animal

studies would indicate. A variety of examples of such evidence may be cited.[10,11] Perhaps, the best known is the incident that occurred in Seveso, Italy, in 1976, where thousands of people were exposed to high doses of dioxins as a result of a chemical plant explosion. Subsequent testing of Seveso residents revealed dioxin levels as high as 56,000 parts per trillion - 10,000 times higher than the 5 to 6 ppt normally found in human tissue. In spite of these high levels, epidemiological studies showed no increase in health problems in Seveso residents 15 years after the incident. A recent study of more than 5,000 chemical workers exposed to high levels of dioxin did not detect any statistically significant differences in cancer mortality between this group and the general population. Exposures of large numbers of people to high dioxin levels at Nitro, West Virginia, in 1949, and at Times Beach, Missouri, in 1982, have similarly failed to result in identifiable serious health problems.

The reasons for the sharp disparity between the results of epidemiological studies of humans and the predictions based on animal testing can only be speculated upon. It should be noted, however, that there is a wide variation in species of laboratory animals with respect to their susceptibility to the toxic effects of dioxin, and that extrapolation of the data for any given species to humans can give very different results, depending on the toxicological model used to interpret the experimental data. The key difference concerns the disputed existence of a threshold level below which dioxin is not toxic.[12] The epidemiological evidence discussed above provides support for the assumption of a threshold mechanism.

In the face of these grave uncertainties concerning the toxicity of dioxin, it is difficult to apply the simple regulatory model referred to above. Nevertheless, in the absence of suitable alternatives, regulating agencies have chosen to employ it to estimate allowable exposures for assumed risk levels. Comparison of the estimates made by various agencies is one manifestation of the degree of uncertainty surrounding the issue. As shown in **Table II**, these vary by several orders of magnitude, with the estimate made by the USEPA being the most conservative.

Measurements made at an early phase of the industry's involvement in efforts to limit its dioxin releases indicated that the median 2378-TCDD and 2378-TCDF contents of bleached pulps were respectively 4 and 18 ppt. Values measured in paper products at that time were of the same order of magnitude as those measured in bleached pulps. To evaluate the implications of these levels NCASI performed risk assessment analyses for dioxins in printing and writing

papers, personal care products, and food contact products[12]. Using the USFDA estimate of carcinogenic potency, these studies showed that to pose an incremental risk of 1 in 1,000,000, the dioxin concentration in printing and writing papers and personal care products would have to be orders of magnitude higher than observed levels. The corresponding concentration in coffee filters for the upper 90th percentile consumer was higher than the highest value measured in bleached paper coffee filters. For food contact papers in general, the results of NCASI migration studies were transmitted to the USFDA for use in its risk assessment. Although these risks were determined to be low, the industry entered into a voluntary commitment that limits the dioxin content of all food contact products made from bleached pulp.

Table II. 2378-TCDD Dose Required for Incremental Cancer Risk of 1 in 1,000,000 as Estimated by Various Government Agencies[12]

COUNTRY	AGENCY	RISK-SPECIFIC DOSE (pg/kg-day)[a]
USA	Environmental Protection Agency	6.4×10^{-3}
USA	Centers for Disease Control	2.8×10^{-2}
USA	Food and Drug Administration	5.7×10^{-2}
CANADA	National Research Council	6.5×10^{-2}
GERMANY		1.0
USA	New York State	2.0
NETHERLANDS		4.0
CANADA	Health and Welfare Canada	10.0

[a]Picograms (10^{-12}g) per kg body weight per day

In the United States, the USEPA has issued a guideline to the state governments for their use in setting dioxin limits. The guideline specifies an ambient water quality standard of 0.013 parts per quadrillion 2378-TCDD in the receiving stream. This is based on the conservative USEPA risk-specific dose assumption shown in **Table II**. That agency is currently re-examining this assumption but has announced no plans to amend its dioxin guideline.

PULP BLEACHING AS A SOURCE OF DIOXINS

Following the initial screening study[2] of five representative mills, a second study was undertaken by the industry in cooperation with the USEPA.[3] This study consisted of a sampling program that encompassed all 104 mills that practiced chlorine-based bleaching in April, 1988. At each mill the three export vectors (pulp, effluent, and sludge) were analyzed for 2378-TCDD and 2378-TCDF. The results, as recorded in a recent NCASI report of the status of the industry's dioxin control effort,[13] are shown in **Table III**. It is apparent that dioxins discharge from bleached pulp mills at that time was a widespread phenomenon.

Table III. Results of 104 Mill Study[13]

	EFFLUENT		SLUDGE		PULP	
	TCDD ppq	TCDF ppq	TCDD ppt	TCDF ppt	TCDD ppt	TCDF ppt
MAX	640	8400	1390	17100	116	2620
90th %ile	110	900	161	1300	22	157
MEAN	51	443	72	607	8.7	91.5
MEDIAN	21	68	16	78	4.4	18

Mechanism of Formation

Laboratory studies were initiated shortly after the industry became aware of the dioxin issue. Among the early results was the demonstration by Swedish researchers[14,15] of the strong link between the amount of chlorine applied and the amount of dioxin formed. Subsequent research at the Pulp and Paper Research Institute of Canada[16] offered an explanation in the form of a hypothesis that TCDD and TCDF are formed by the reaction of chlorine with DBD and DBF, the unchlorinated analogs, by simple aromatic substitution reactions in which the hydrogen atoms of DBD and DBF are progressively replaced by chlorine atoms. This model assumes that DBD and DBF are present in the unbleached pulp and react with any chlorine that remains after the rapid initial phase of lignin chlorination is complete.

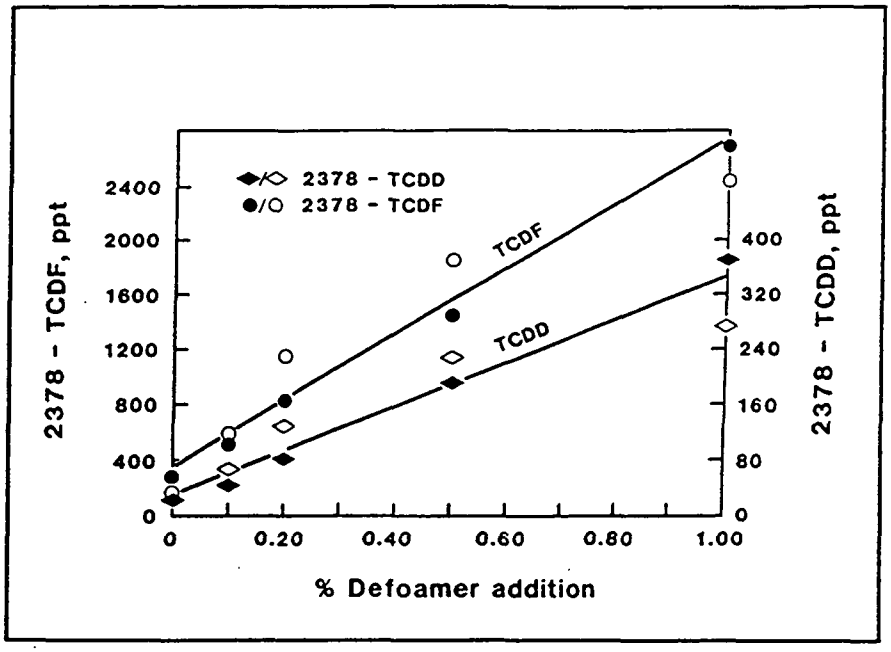

Figure 2. Dioxins Formation in Duplicate Chlorination Experiments as a Function of Defoamer Addition to Western Hemlock Kraft Brownstock.[18]

A variety of observations were made that lend credence to the theory that the dioxins formed during bleaching originate by substitution of chlorine into DBD and DBF. Canadian researchers[17,18] showed that oil-based defoamers, because of contamination with DBF, and probably also DBD, contribute to the formation of chlorinated dioxins during bleaching. Linear increases in the TCDF and TCDD concentrations in chlorinated pulp were observed as the amount of defoamer added to the brownstock was increased, as shown in **Figure 2**. Defoamers containing nondetectable amounts of DBF, on the other hand, had no significant effect on the amount of TCDD and TCDF generated during chlorination.

Additional evidence for the involvement of DBD and DBF came in the form of findings that (1) DBD and DBF are present in typical brownstocks;[19] (2) addition of DBD and DBF to brownstock before chlorination gives elevated levels of TCDD and TCDF, the incremental TCDD and TCDF being similar in isomer distribution to the TCDD and TCDF formed from the brownstock to which no DBD and DBF had been added;[16,18,20] (3) solvent extraction to remove DBD and DBF from the brownstock before chlorination sharply decreases the formation of TCDD and TCDF;[16,20] (4) TCDD and TCDF are formed only in the chlorination stage, not in the extraction stage, as would be

more likely if, for example, chlorophenols were the precursors;[20,21,22] (5) dioxins formation increases with increasing kappa factor in a way that is consistent with DBD and DBF involvement;[16] and (6) model chlorination experiments show that mono-, di-, and trichlorinated dioxins are formed en route to formation of the tetrachlorinated ones.[23]

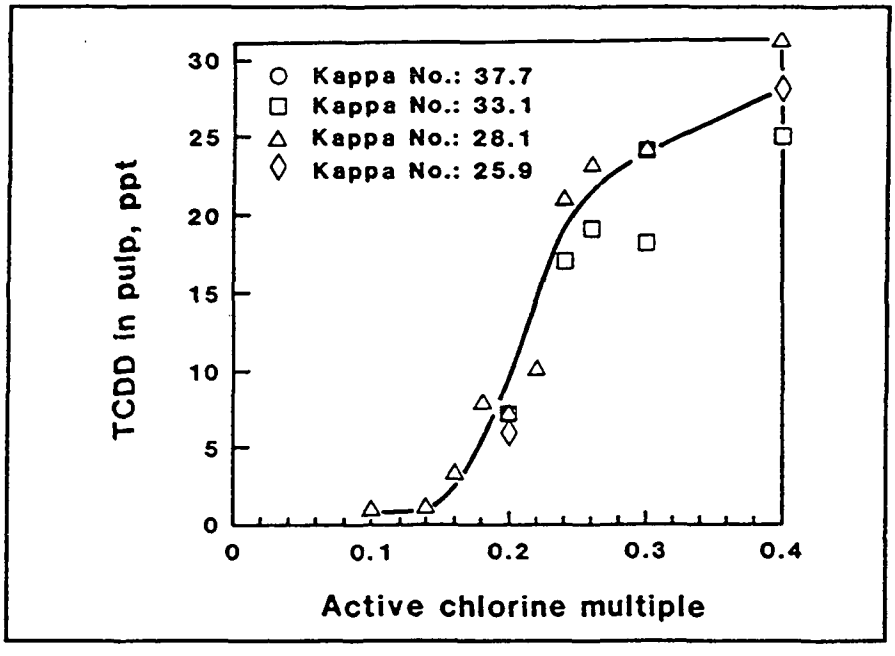

Figure 3. TCDD Formation as a Function of Chlorine Charge.[16]

Figure 3 shows the dependence of TCDD formation on the ratio of chlorine charge (% o.d. pulp wt.) to kappa number, here referred to as active chlorine multiple, in the absence of chlorine dioxide.[16] The shape of the curve may be easily rationalized on the basis of the direct DBD chlorination hypothesis if it is assumed that lignin is significantly more reactive than DBD toward chlorine. Thus, nondetectable or very small amounts of TCDD are formed at low chlorine charges because all of the chlorine added is consumed by lignin before it has an opportunity to react with DBD. As the chlorine charge is raised beyond a critical threshold value corresponding to a chlorine multiple of about 0.15, sufficient chlorine remains after the initial rapid reaction with lignin to convert part of the DBD to TCDD. The extent of this conversion continues to increase with increasing chlorine charge until a point is reached at which conversion of TCDD into pentachlorinated isomers begins to compete favorably with its formation.

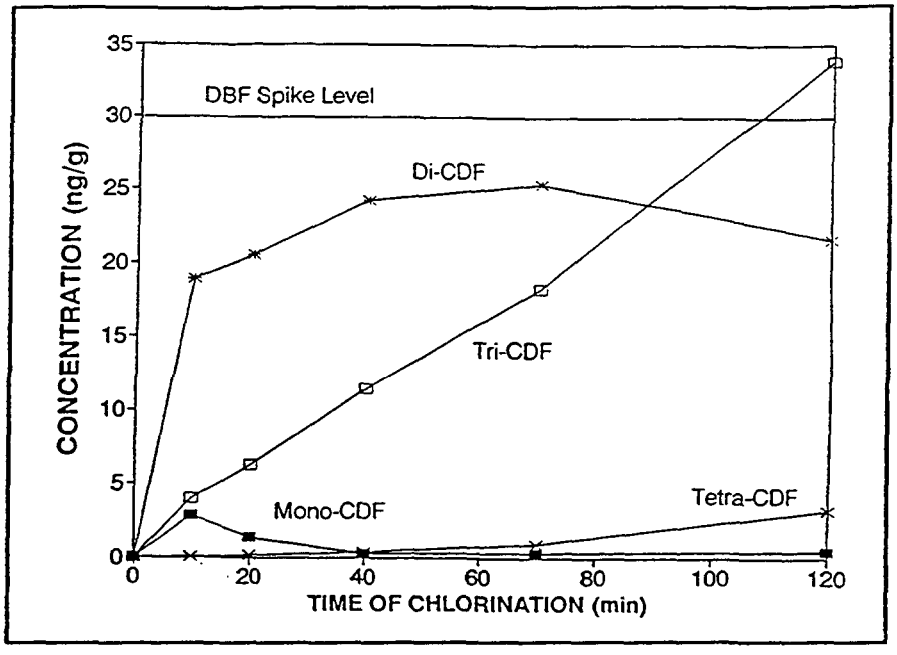

Figure 4. CDF Formation on Chlorination of DBF-spiked Cotton Linters.[23]

Further evidence for the direct substitution mechanism was obtained in model studies by Dimmel and co-workers.[23] **Figure 4** shows the progressive development of ring chlorination of DBF adsorbed by cotton linters. The development of the various congeners with time is as expected for the progressive DBF chlorination mechanism. Mono-CDF formation was also observed to pass through a maximum when solvent-extracted kraft pulp was chlorinated, indicating that some DBF remained after extraction; no direct determination of residual DBF was made.

In spite of this apparently overwhelming evidence for the direct chlorination hypothesis for dioxins formation during bleaching, there are persistent indications that other precursors may make a significant contribution. The NCASI intensive study[21] demonstrated a remarkable lack of correlation between brownstock DBD concentration and TCDD formation in (admittedly less well-controlled) mill environments, as shown in **Figure 5.**

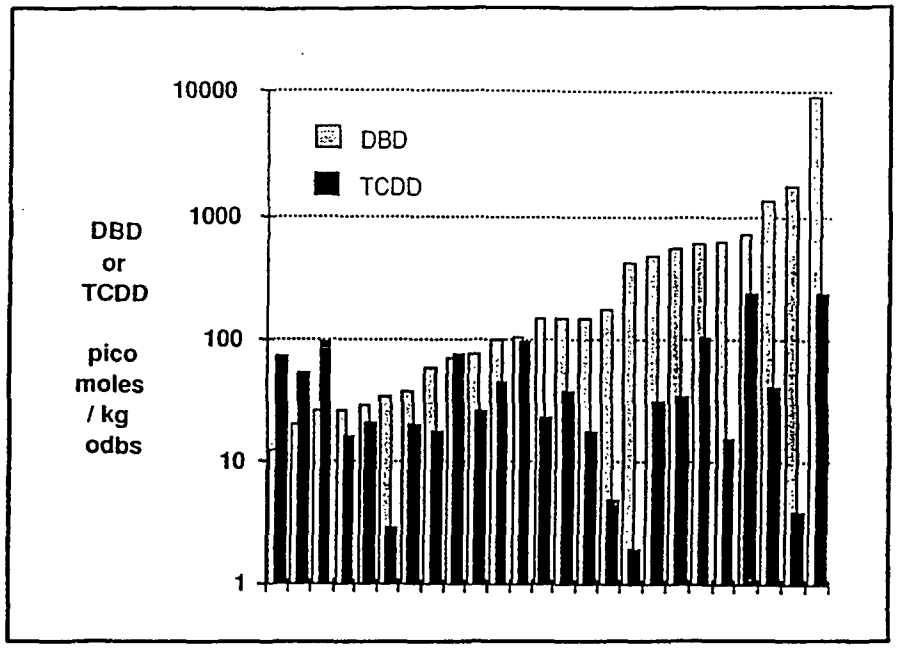

Figure 5. Comparison of Brownstock DBD Levels with Levels of 2378-TCDD Generated in Bleach Plants.[22]

Several research groups have attempted to investigate the possibility of other precursors by removing DBD and DBF from the brownstock prior to chlorination.[8,20,24] In one of these studies,[20] particularly stringent extraction conditions were used, and the extraction was followed by steam distillation to ensure that all extractable precursors were removed. Subsequent analysis confirmed that no more than 0.6 ppt DBD and only 49 ppt DBF remained after extraction. In spite of these low levels, the chlorinated pulp was found to contain 20 ppt 2378-TCDD, 38 ppt 2378-TCDF, and 87 ppt of other tetra- through hepta-CDFs. These results demonstrate the existence of nonextractable precursors. Possible explanations include the presence of DBD and DBF moieties which are strongly bound, either physically or chemically, to some component of the pulp fiber wall, or formation of DBD from residual lignin during chlorination. These hypotheses gain support from the work of Hrutfiord and Negri,[25] who showed that synthetic lignin of the p-hydroxybenzyl type yields high concentrations of TCDD and TCDF upon chlorination. Since lignin of this type is found in softwood compression wood (such as occurs in and around knots, and on the underside of branches and leaning stems), compression wood and pulps made from it were chlorinated and analyzed. They exhibited much higher TCDD and TCDF levels than the corresponding normal wood and pulp. Additional support is provided by the results of Hise *et al.*,[26] who showed that chlorination of lignin produces dioxins which are similar in amount and isomer composition to those produced by chlorinating kraft pulp.

Other possible precursors for dioxins are chlorophenols of the type known to be produced during chlorination and polychlorinated phenols sometimes used as wood preservatives by sawmills who are also suppliers of chips for pulping. Model experiments[26] have shown that chlorinated phenols of the former type yield dioxins under pulp chlorination conditions, but the isomer distribution patterns are not typical of dioxins formed during pulp chlorination. An investigation of the latter possibility has shown that the polychlorinated phenol wood preservatives are contaminated with chlorinated phenoxyphenols that are converted to hexachlorinated dioxins and furans in the digester.[27]

PREVENTION OF DIOXIN FORMATION DURING BLEACHING

It is clear from the foregoing discussion that the research effort undertaken by the industry when the dioxin problem was discovered has been massive and largely successful. The magnitude of the problem was quickly delineated; the location of formation was determined; and likely mechanisms were identified. Furthermore, the information obtained has been used to develop an array of methods for preventing the generation of detectable quantities of dioxins.

These methods are all based on the simple mechanistic model of chlorine reacting with precursors in the chlorination stage. This model suggests two general approaches to prevention: reduction of the concentration of chlorine and reduction of the concentration of precursors.

Control Strategies Based on Precursor Reduction

The precursors DBD and DBF have been strongly implicated, and the goal of minimizing their concentration in the pulp entering the chlorination stage is therefore an important one. It may be achieved through improved brownstock washing,[24] judicious selection of any defoamers[18] used in the pulp washing system, appropriate choice of the liquid to be used in the showers on the washer preceding the chlorination stage,[28,29] use of oxygen predelignification,[8,30] and appropriate control of chlorination stage filtrate recycle.[14,28] Methods of chemically destroying precursors are still at the research stage, but may become available in the not-too-distant future.[31,32]

Oxygen predelignification has been shown to be effective by virtue of the opportunity it provides for steam stripping of precursors from the pulp.[18,33] It was among several control

methods evaluated in a systematic laboratory study,[30] together with delayed chlorine dioxide addition, increased chlorination temperature, elimination of chlorination stage filtrate recycle and improved brownstock washing, and was found to be the most effective of these methods.

Chemical destruction of precursors can be effected by ozone, nitrogen dioxide, hydrogen peroxide, nitric acid, or hydroxyl radicals formed by ultraviolet radiation.[31,32] The effect of ozone on precursors dissolved in water is shown in **Figure 6**. Of the reagents tested, ozone appeared the most promising, but it is not yet clear whether the precursors would compete successfully with the lignin in the unbleached pulp for the available ozone at charges sufficiently low for the ozone stage to be considered a pretreatment, rather than a replacement for chlorine.

The model studies already described[25] suggest that reaction wood should be avoided and that knots should be removed before the wood chips are fed to the digester. To the extent that normal lignin is a precursor, as suggested by several laboratory studies,[20,26] the use of extended delignification is suggested.

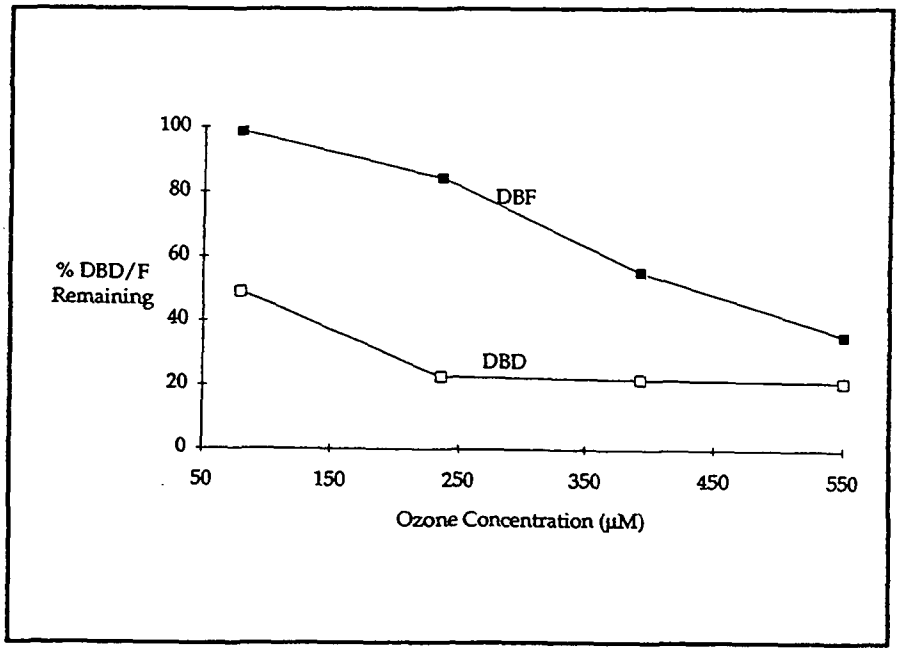

Figure 6. Reactions of Ozone with DBD and DBF in Unbuffered Solutions. DBD and DBF were initially present at about 3 ppm.[31]

Control Strategies Based on Chlorine Reduction

Among the earliest findings of the relevant research was the critical importance of the ratio of chlorine charge to kappa number (chlorine factor). As seen in **Figure 3**, when the chlorine factor is below 0.15, the resulting TCDD levels are nondetectable or nearly so. This decrease is most readily achieved by substituting chlorine dioxide for part of the chlorine. The effect is shown in **Figure 7**. As the substitution level is increased at a constant active chlorine multiple (kappa factor, [Cl_2 charge + 2.63 x ClO_2 charge], % o.d. pulp wt./kappa no.), dioxin production decreases to a value of zero at some critical substitution level. It is also apparent that the order of addition of chlorine and chlorine dioxide is important. If the substitution level is sufficiently high and the chlorine is added first, it is rapidly consumed by lignin, and none remains to form dioxin. If the chlorine dioxide is added first, it rapidly satisfies much of the lignin's bleach demand, so that when the chlorine is added it remains at a high enough concentration to react with dioxin precursors. It is important to remember, however, that at a given kappa factor and substitution level, adding chlorine dioxide first results in more efficient delignification than adding chlorine first. Consequently, if chlorine is added first, the kappa factor must be increased, with the result that dioxins formation increases to the extent that no net reduction in dioxin levels is achieved.[34]

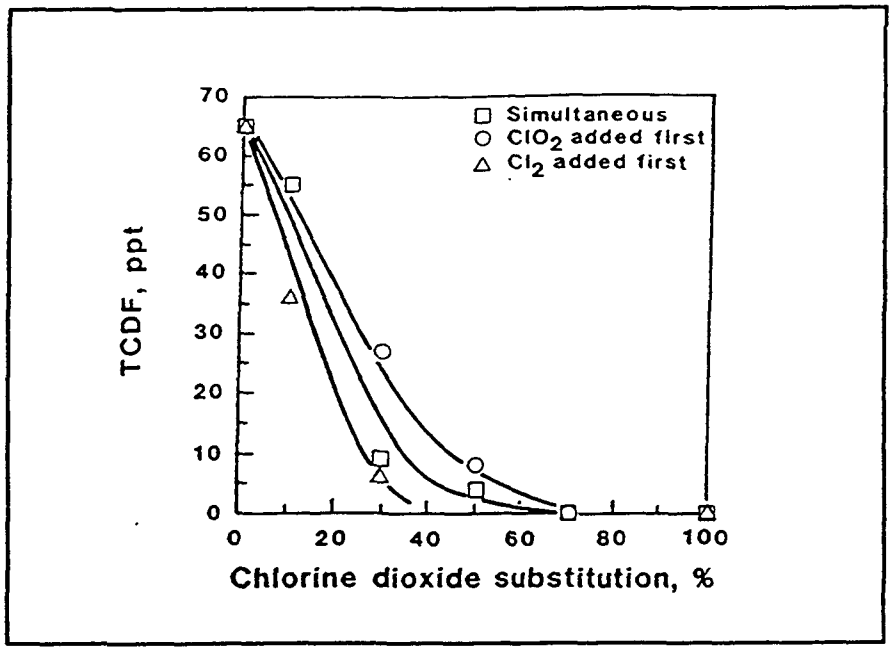

Figure 7. Effect of Chlorine Dioxide Substitution and Order of Addition on TCDF Formation at a Kappa Factor of 0.21.[16]

Returning to the effect of substitution, Canadian researchers[16] carried out experiments of the type shown in **Figure 7** at various kappa factors to determine the substitution level required to reduce the dioxins to nondetectable levels. This was plotted against kappa factor to give the line shown in **Figure 8** (in which kappa factor is identified by its synonym, active chlorine multiple). The filled data points denote experimental or mill operating conditions that produced measurable dioxin levels. The unfilled ones denote nondetectable dioxins. Operation at conditions to the left of or above the line may be expected to produce no detectable dioxins.

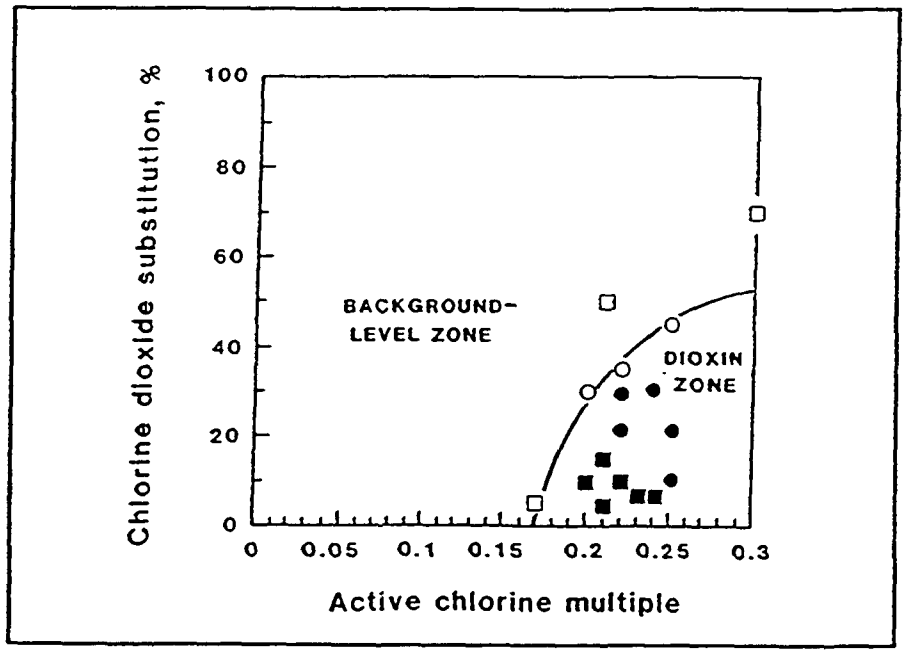

Figure 8. Conditions of Kappa Factor and ClO_2 Substitution that Promote (lower right) or Inhibit (upper left) Dioxins Formation. Filled Data Points Denote Measurable Dioxins Levels.[16]

Researchers at Westvaco Corporation have developed another method of avoiding high chlorine concentrations.[35] It consists of dividing the total chlorine charge into several smaller charges that are added separately. This has been shown to be effective in reducing dioxins levels. In a similar vein, good mixing and good control of the rate of addition of chlorine may be expected to reduce both chlorine concentration and dioxin levels.

Other methods for reducing chlorine concentration exploit the equilibrium between molecular chlorine and hypochlorous acid:

$$Cl_2 + H_2O = HOCl + H^+ + Cl^-$$

This equilibrium can be displaced to the right, reducing molecular chlorine concentration and decreasing the ratio of Cl_2 to HOCl by (1) adding water (reducing consistency), (2) reducing the concentration of H^+ (increasing pH), (3) reducing chloride ion concentration, and (4) increasing the temperature (because the equilibrium constant increases with temperature). Increasing pH by base addition has been shown to reduce dioxins formation,[35] but must be practiced with caution because the pulp strength can be adversely affected. Increasing chlorination temperature from 30 to 60°C has been observed to decrease TCDD and TCDF formation by 50%.[30] Chloride ion concentration can be kept low by avoiding chlorination stage filtrate recycle.

THE STATUS OF DIOXIN ABATEMENT IN THE PULP AND PAPER INDUSTRY

The results of the large volume of dioxins research conducted by the industry have been speedily implemented. **Table IV**[13] compares 1992 levels in the U.S. industry with those of the 104 Mill Study. The reductions are close to 90% on a TEQ basis.

Table IV. Comparison of 1988 and 1992 Dioxins in U.S. Bleached Pulp Mills.[13]

Year		EFFLUENT		SLUDGE		PULP	
		TCDD ppq	TCDF ppq	TCDD ppt	TCDF ppt	TCDD ppt	TCDF ppt
1988	MAX	640	8400	1390	17100	116	2620
	90th %ile	110	900	161	1300	22	157
	MEAN	51	443	72	607	8.7	91.5
	MEDIAN	21	68	16	78	4.4	18
1992	MAX	80	510	133	735	10	323
	90th %ile	10	65	32	110	2.6	10
	MEAN	6	31	11	55	0.9	6
	MEDIAN	ND(4)	6	2	11	0.5	0.6

Similar information is provided for the Canadian industry in **Figure 9.** A reduction of approximately 95% has been achieved.

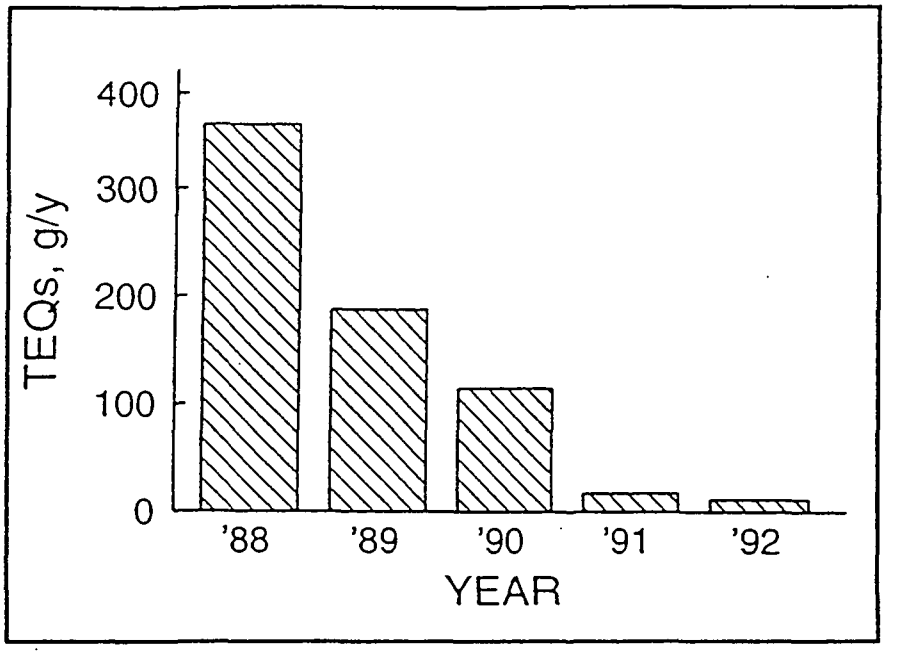

Figure 9. 2378-TCDD and TCDF TEQ Discharges by the Canadian Pulp and Paper Industry, 1988-1992.[36]

SUMMARY AND CONCLUSIONS

Polychlorinated dibenzodioxins (PCDDs) and dibenzofurans (PCDFs) include compounds that are extremely water insoluble, lipophilic, strongly sorbed, very stable, and highly toxic toward laboratory animals. This combination of properties makes PCDDs and PCDFs highly objectionable from an environmental standpoint. Consequently, their discovery in pulps, effluents, and sludges from bleached pulp mills sparked a massive research effort by the pulp and paper industry to identify formation mechanisms and control strategies.

This research was complicated by the extreme smallness of the amounts present in pulp mill export vectors - a few parts per trillion in pulps and sludges, parts per quadrillion in effluents. The necessary analytical methodology has been developed in recent years. It employs extensive analyte enrichment procedures and quantification by isotope dilution gas chromatography/mass spectroscopy.

This research has been successful. The chlorination stage of pulp bleaching was identified as the source, the unchlorinated analogs as important precursors, and direct substitution of chlorine as an important mechanism of formation. This information has been used to develop a variety of control strategies. Some of these are directed toward reducing the amounts of precursors entering the chlorination stage, some toward reducing the amount of chlorine used. Their implementation has resulted in a reduction of more than 90% in the amount of dioxins released by North American bleached pulp mills.

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