

# Effect of Gaseous Inhibitors on PCDD/F Formation

PÄIVI H. RUOKOJÄRVI,<sup>†</sup>  
 ISMO A. HALONEN,<sup>†</sup>  
 KARI A. TUPPURAINEN,<sup>‡</sup>  
 JUHANI TARHANEN,<sup>†</sup> AND  
 JUHANI RUUSKANEN<sup>\*†</sup>

Department of Environmental Sciences and  
 Department of Chemistry, University of Kuopio,  
 P.O. Box 1627, FIN-70211 Kuopio, Finland

Emissions of polychlorinated dibenzo-*p*-dioxins (PCDDs) and dibenzofurans (PCDFs) from municipal waste incineration are currently a subject of considerable public concern because of their extreme toxicity. PCDD/F formation in incineration processes is being studied widely, but studies on inhibition are quite sparse, especially in a pilot-plant scale. In this work, the effect of four gaseous inhibitors (sulfur dioxide, ammonia, dimethylamine, and methyl mercaptan) on PCDD/PCDF formation in the combustion of liquid fuel was studied using a pilot-scale plant. The inhibitors were injected into the flue gas stream after the first economizer at a temperature of 670 °C and just before the second economizer at 410 °C. Both the chlorophenol and PCDD and PCDF concentrations decreased when inhibitors were added. Particle-phase PCDD/F concentrations in particular decreased by up to 98%. The results suggest that the formation of PCDD/Fs is hindered in the particle phase at the early stages of the PCDD/F formation chain, probably even before precursors such as chlorophenols have been formed.

## Introduction

Incineration is a widely used method for disposing of waste. Unfortunately, it is also a major source of toxic PCDD/F emissions in our environment. Perhaps over 30% of all dioxins are formed during municipal solid waste (MSW) combustion (1). PCDD/Fs were first discovered in MSW incinerators in 1977 (2), and it has since been confirmed on several occasions that their formation in the course of incineration mainly takes place in the postcombustion zone, involving various catalyzed mechanisms. Among others, our previous investigations have corroborated the importance of both chlorine and metal catalysts (especially Cu<sup>2+</sup>) for PCDD/F formation (3, 4). Comprehensive summaries of the reaction pathways involved in PCDD/F formation have recently been presented by Addink and Olie (5) and Froese and Hutzinger (6, 7).

PCDD/F emissions from flue gases can, of course, be controlled via the composition of the fuel and through various aspects of combustion process technology, e.g., the temperature in the furnace and postfurnace sections, turbulence and residence time, and by means of flue gas cleaning systems

such as electrostatic precipitators (ESP) and fabric filters. Both these devices are successfully used to decrease PCDD/F emissions (8), but in some experiments ESPs have also been observed even to increase these emissions (9–13). PCDD/F destruction was also noticed to differ between the different congeners (14), and also shifts in the congeners are possible when flue gases pass by the ESP (15). The combinations of dry scrubber/fabric filter (16), wet scrubber/fabric filter (17, 18), or spray absorber/ESP (19) and wet gas scrubbing (20) have also been used successfully to decrease PCDD/F emissions. PCDD/F reduction accompanied with NO<sub>x</sub> reduction by ammonia injection (21) or ammonia radical injection and by using SCR unit or some other DENOX unit are well-known methods for PCDD/F reduction (22–25). The injection of different sorbents and absorption materials to the flue gases are also potential methods for reducing the emissions of PCDD/Fs and related compounds. For example, water spray injection acts as an absorption material and also cools the flue gases to the temperature, which does not favor PCDD/F formation and thus may lead to decreases in PCDD/F emissions (26). Water injection was observed to reduce the gas-phase PCDD/Fs and highly chlorinated species more effectively than particle-phase PCDD/Fs or tetra to penta congeners, which were noticed even to increase with water injection (27). In general, several flue gas cleaning devices are used simultaneously to reduce PCDD/Fs and other flue gas component emissions.

The best way, however, is to block the formation of PCDD/Fs completely, thus avoiding the problems of dioxin formation and the presence of these substances in fly ash. This may turn out to be difficult in practice, however. Accordingly, various inhibitors have been tested both in the laboratory and in pilot plants (28–35). There is clear evidence that some compounds actually reduce PCDD/Fs in flue gases, although no final solution has been found nor has the mechanism of inhibition been completely resolved. In particular, the role of sulfur and nitrogen additives has been discussed with respect to reducing PCDD/F formation. The catalytic formation of dioxins on particulate surfaces in waste incinerators can be inhibited by treating the particulates with sulfur-containing compounds (28, 33, 35), ethanalamine (29, 31), or triethanolamine (29, 30). In view of these results, which are for the most part based on the laboratory-scale studies, we performed a series of experiments to determine whether injecting related gaseous compounds directly into the flue gases would significantly reduce PCDD/F formation in a pilot-plant scale. The aim here is to describe the reduction of PCDD/F formation in a pilot plant using different gaseous compounds as inhibitors. Although the details of the inhibition mechanisms cannot be firmly established, it is our hope that the results will contribute to the development of new inhibitors to cope with the problem of dioxin formation.

## Experimental Section

The tests were carried out in a 50-kW pilot plant (Figure 1) consisting of an oil burner and a furnace from which the flue gases were directed through two economizers to the stack. Sampling was performed isokinetically after the second economizer.

The basic fuel used in the tests was light heating oil. Chlorine, in the form of tetrachloroethylene, and copper (catalyst) as copper nitrate were added to the fuel and adjusted to correspond to 0.5% of the total fuel flow (by mass). These additives (p.a., purchased from Merck) were selected according to our previous studies (3, 4) and were used to

\* To whom all correspondence should be addressed. Telephone: +358-17-163227; fax: +358-17-163230; e-mail: Juhani.Ruuskanen@uku.fi.

<sup>†</sup> Department of Environmental Sciences.

<sup>‡</sup> Department of Chemistry.

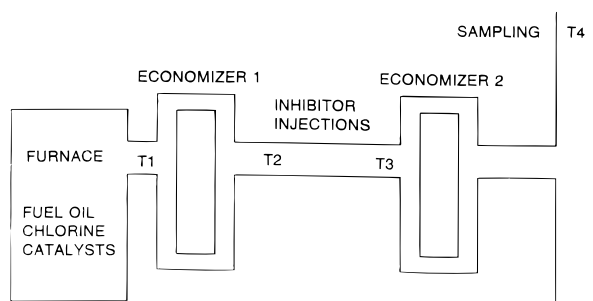


FIGURE 1. Scheme of the pilot plant used in the inhibitor tests.

increase the PCDD/F formation in the tests to the level corresponding MSW incinerator emissions. The four gaseous inhibitors tested were sulfur dioxide ( $\text{SO}_2$ ), ammonia ( $\text{NH}_3$ ), dimethylamine (DMA), and methyl mercaptan (MM), all purchased from AGA, with a purity > 99.0%. These inhibitors were sprayed into the flue gases at two locations (see Figure 1), after the first economizer at T2 ( $T = 670^\circ\text{C}$ , inhibitor concentrations 100 or 200 ppm in flue gas) and immediately before the second economizer at T3 ( $T = 410^\circ\text{C}$ , concentration 100 ppm in flue gas). Methyl mercaptan was injected only at T3. Blank tests were carried out before each run to check the absence of "memory effects".

Chlorophenol and PCDD/F samples were collected in a glass fiber filter (Schleicher & Schuell) at location T4 for particulate phase (kept in a box at  $120^\circ\text{C}$  during the sampling period to prevent water condensing onto the filter) and in XAD-2 resin (Amberlite, 20–50 mesh) for gas phase after removing the condensation water from the sample stream. The particulate mass collected on the filter was measured by weighting the filter before and after the sampling at constant temperature and relative humidity ( $20^\circ\text{C}$  and 45%, respectively).

The following flue gas parameters were analyzed continuously at the same location (T4):  $\text{O}_2$ , CO,  $\text{SO}_2$ ,  $\text{C}_x\text{H}_y$ , and  $\text{NO}_x$ . HCl concentrations in the flue gas were analyzed three times during each test by absorption into a 0.1 M NaOH solution.

Details of the analytical procedure have been described elsewhere (36, 37). It consists of Soxhlet extraction with toluene, chlorophenol extraction with potassium carbonate, and PCDD/F purification with concentrated sulfuric acid and multistep columns. The particulate and gaseous phases were analyzed separately. Finally, the samples were analyzed using a HP 5890 gas chromatograph with a HP 5970 mass selective detector (chlorophenols) and by a HRGC/HRMS (VG 70 250 SE), operating the MS at a resolution of 10000 (PCDD/Fs).

## Results and Discussion

The flue gas parameters during the test runs are presented in Table 1. The flue gas temperatures at different locations in the pilot plant (mean values) were as follows: T1 ( $870^\circ\text{C}$ ), T2 ( $670^\circ\text{C}$ ), T3 ( $410^\circ\text{C}$ ), and T4 ( $250^\circ\text{C}$ ). HCl concentrations were  $770\text{ mg/m}^3$  (no inhibitor),  $290\text{ mg/m}^3$  ( $\text{SO}_2$  injection at  $410^\circ\text{C}$ ), and  $190\text{ mg/m}^3$  ( $\text{NH}_3$  injection at  $410^\circ\text{C}$ ). Note that CO levels were high throughout the tests. In fact, this behavior is typical for small pilot plants, and moreover, it also guaranteed that there was enough carbon in flue gases for PCDD/F formation.

The results indicate that a clear reduction in both chlorophenol and PCDD/F concentrations occurred when inhibitors were injected into the flue gas stream (Figures 2 and 3) even though the concentrations of inhibitors were quite low, i.e., 100 and 200 ppm.

**Chlorophenol Reductions.** Less than 20% of the chlorophenols were found in the particulate phase throughout

the tests, due to their fairly volatile nature. Also, chlorophenol concentrations reduced slightly more in the particle phase than in the gas phase. The total chlorophenol reductions when inhibitors were used varied from 11 to 53%, but there were two exceptions as  $\text{SO}_2$  and  $\text{NH}_3$  seemed not to have any inhibitory effect on chlorophenol concentrations when injected before the second economizer at  $T = 410^\circ\text{C}$  (Figure 2). The most abundant chlorophenol isomers throughout the tests were 2,4- and 2,5-DCPs, which accounted for approximately 40% of the total chlorophenol concentration, and 2,4,6-TCP, 2,3,4,6-TeCP, and PCP. No significant change in isomeric distributions could be observed when the inhibitors were injected into the flue gas stream.

Among others (38–40), our previous investigations have shown that the amounts chlorobenzenes and chlorophenols correlate with those of PCDD/Fs (41). In this particular study, however, the observed correlation between chlorophenols and PCDD/Fs was only fair. Unfortunately, we have no sound explanation for this behavior.

**PCDD/F Reductions.** Overall PCDD/F concentrations were reduced by 42–78% (Figure 3), the only exception being  $\text{SO}_2$  when injected just before the second economizer at  $T = 410^\circ\text{C}$ , which did not seem to reduce PCDD/F concentrations significantly during the tests (a decrease of about 15% in total PCDD/F concentrations). The particle-phase PCDD/F concentrations decreased by up to 98% (for DMA). No significant reductions in the gas-phase concentrations were observed in the tests except for DMA injected at  $670^\circ\text{C}$ , which resulted a reduction in PCDD/F to 42%. Particle-phase reductions were also calculated from the dioxin concentration per unit particle weight (ng of PCDD, PCDF/mg of particles) to see if the variation in particle concentration had any effect on the reduction, but the reductions per unit particle mass were in general of the same order as those in the particle phase (in ng/m<sup>3</sup>). For the  $\text{SO}_2$  test ( $410^\circ\text{C}$ ), the reduction per particle mass was better than the "absolute" reduction, possibly due to the fact that the greater total particle surface area involved in the formation of PCDD/Fs (which results from the higher particle concentration assuming that particle size distribution remains unchanged) provided more active sites for catalysis, and the injection temperature or the amount of inhibitor were inadequate for successful inhibition in this case.

The earlier the inhibitor was injected and the higher its concentration, the greater was the observed inhibition. The reductions were largest when the inhibitors were injected into the flue gas at  $670^\circ\text{C}$ , as they were able to block PCDD/F formation more completely at the earlier stage (T2), and at this point the residence time before the sampling point is also greater than at point T3. It is therefore important that an inhibitor should be added to the flue gas before PCDD/F formation has occurred, since they do not destroy PCDD/Fs that have already been formed but prevent their formation, probably at an early stage in the chain (6), even before the chlorophenol precursors have been formed. Consequently, when injected immediately after the furnace, these inhibitors are able to block the PCDD/F formation that would normally occur in the postcombustion zone.

We also investigated possible changes in homologue patterns when inhibitors are present. No statistically significant change in PCDD or PCDF homologue (congener class) patterns could be observed in the samples in which inhibitors were used relative to those samples without inhibitors, but a slight indication of the dechlorination of more highly substituted congeners (hepta and octa) could be detected, especially in the case of  $\text{NH}_3$ , DMA, and MM. Hepta- and octachlorinated dioxins and tetra- and octachlorinated furans predominated throughout the tests.

Although there may be some minor disturbing effects in gas/particle partitioning (caused by factors such as gas

TABLE 1. Flue Gas Parameters and Measured Chloro Compounds Concentrations in the Inhibitor Tests (Mean Values)

test run <sup>a</sup>	O <sub>2</sub> (%)	CO (mg/m <sup>3</sup> )	SO <sub>2</sub> (mg/m <sup>3</sup> )	particles (mg/m <sup>3</sup> )	ClPh (μg/m <sup>3</sup> )	PCDD/F (ng/m <sup>3</sup> )	[PCDD]:[PCDF]
no inhibitor (4)	4.4	1100	55	114	3.7	340	0.6
SO <sub>2</sub> , 410 °C, 100 ppm (2)	4.0	1350	115	288	4.2	290	0.4
SO <sub>2</sub> , 670 °C, 100 ppm (2)	4.5	1550	180	195	1.9	170	0.5
SO <sub>2</sub> , 670 °C, 200 ppm (2)	4.9	1650	253	115	2.2	130	0.4
NH <sub>3</sub> , 410 °C, 100 ppm (3)	3.9	1150	19	238	3.7	150	0.2
NH <sub>3</sub> , 670 °C, 100 ppm (2)	5.4	1250	88	161	1.7	140	0.5
NH <sub>3</sub> , 670 °C, 200 ppm (2)	5.8	1250	25	132	3.3	110	0.5
DMA, 410 °C, 100 ppm (2)	4.0	1250	30	234	2.3	150	0.3
DMA, 670 °C, 100 ppm (2)	4.8	1550	55	106	2.2	75	0.4
MM, 410 °C, 100 ppm (2)	5.0	1100	131	80	1.9	180	0.4

<sup>a</sup> The number of parallel tests is given in parentheses.

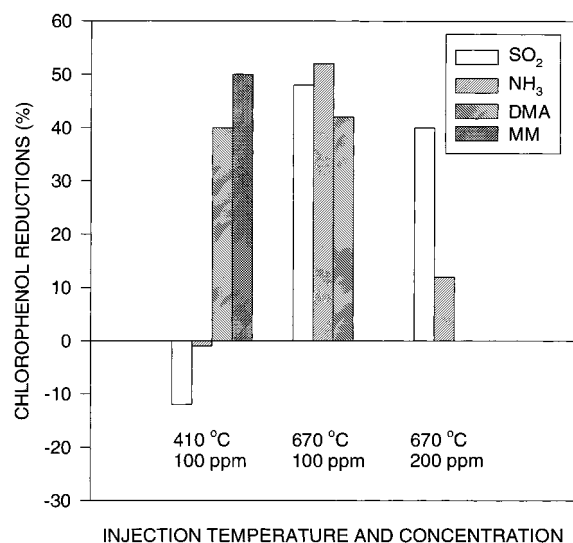
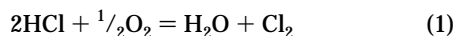


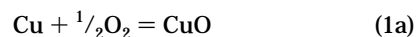
FIGURE 2. Chlorophenol reductions in the inhibitor tests.

molecules collecting on filters, volatile compounds on particles that "blow off" the filters, etc.), we studied whether this partitioning was affected by the use of inhibitors. A clear, albeit difficult to interpret change in the particle/gas distribution can be seen in those cases where inhibitors were used. In the NH<sub>3</sub>, DMA, and MM tests (at both 410 and 670 °C) and in the SO<sub>2</sub> test (at 670 °C, 200 ppm) the amounts of PCDD/Fs in the particulate phase decreased, i.e., inhibitors greatly affected particulate phase formation, and it was only in the case of SO<sub>2</sub> injected at 410 °C that gas/particle distribution remained unaltered. It is evident that the inhibitors function mainly on the particle phase, probably because gas-phase PCDD/F formation is such a rapid process that it has already taken place by the locations at which the inhibitors were injected. The exact impact point cannot be predicted here, as the inhibitors may take effect either by blocking biaryl synthesis (Ullmann reactions) or even by blocking the formation of chlorinated aromatics from aliphatic compounds.

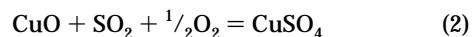
Concerning possible inhibition mechanisms, it seems very likely that the inhibition is, in one way or another, based on the interactions between catalysts and inhibitors. In particular, it is well-known that fly ash can catalyze a reaction between oxygen and hydrogen chloride, giving molecular chlorine or chlorine radicals via the Deacon reaction (42):



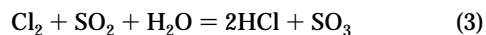
Many copper catalysts (e.g., Cu, CuCl, CuCl<sub>2</sub>, CuO, and Cu<sub>2</sub>O) can promote this reaction considerably (33). Note that the Deacon reaction (eq 1) for chlorine production is probably a two-step mechanism (43):



Moreover, the actual reactions depend on the nature of the Cu catalysts. Consequently, a possible role of sulfur is to react with the Deacon reaction catalysts, e.g., with CuO



to alter their ability to produce Cl<sub>2</sub>. The second postulated role of sulfur is to undergo the following reaction:



thereby converting the effective chlorinating agent (Cl<sub>2</sub>) into a form (HCl) that is less likely to undergo aromatic substitution reactions to produce PCDD/Fs or their precursors. The basic thermodynamics of reactions 1–3 are given elsewhere (44). It seems evident that both Deacon reaction and reaction 3 are thermodynamically favored in the critical temperature range (250–600 °C), yet the inhibition is clearly predominated.

It has recently been observed that the amounts of some metals that are volatilized, particularly copper, depend on the reduction conditions and the amount of chlorine present (45). The presence of sulfur stabilizes many of the metals in sulfate phases at low temperatures but has limited influence above 800 °C (cf. reaction 2). Whether this behavior could provide new inhibition mechanisms remains to be seen.

The third possibility is that sulfur may affect PCDD/F formation by sulfonating phenolic precursors, thus preventing chlorination and Ullmann reactions, or perhaps by forming the sulfur analogues of PCDD/Fs, dibenzothianthrene, and dibenzothiophene, respectively. In any case, it has been suggested that the presence of sulfur in MWIs can reduce the formation of PCDD/Fs and thus co-firing municipal solid waste with coal containing sulfur may reduce PCDD/F emissions considerably (33, 35).

In general, it seems that it is possible to use catalyst inhibitors, i.e., compounds that are strongly absorbed and react with active sites on the catalytic fly ash surface to form stable, inactive complexes with metallic compounds, thereby reducing or eliminating the catalytic activity of the metals or their oxides. All the molecules with lone pair electrons, e.g., those containing nitrogen or sulfur, are promising in this respect as they can easily form stable complexes with copper, iron, and other transition metals (46). In addition, it has been proposed that various alkaline chemicals can suppress PCDD/F formation. The addition of limestone or calcium oxides (47) prevents HCl decomposition, for example, and ammonia behaves similarly (48). The best results have been obtained with organic amines (31), e.g., ethanolamines, which



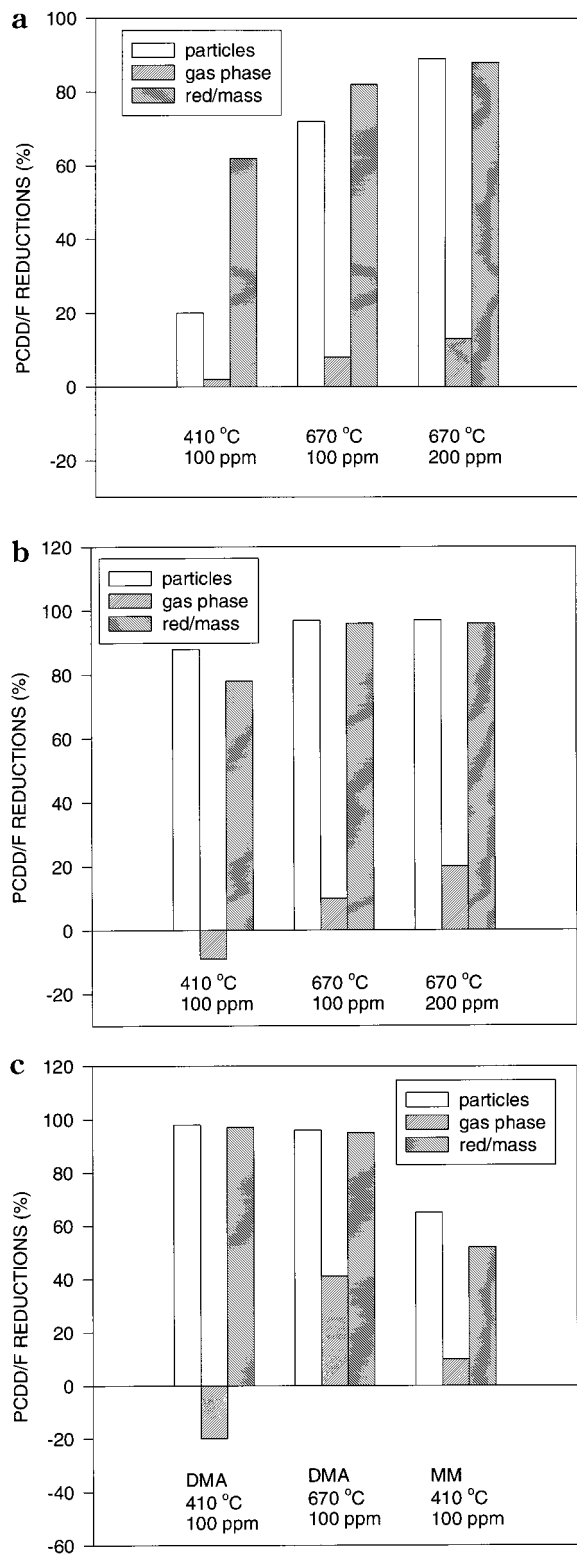


FIGURE 3. PCDD/F reductions in inhibitor tests [calculated from PCDD/F concentrations in flue gas ( $\text{ng}/\text{m}^3$ ) = particles and gas phase and with PCDD/F concentrations per unit particle mass ( $\text{ng}/\text{mg}$  particles) = red/mass]. (a) Sulfur dioxide injection. (b) Ammonia injection. (c) DMA and MM injections.

have been used to block active sites, probably through the formation of copper nitrides, but from a practical point of view, molecules containing amino nitrogen (particularly that derived from biogenic waste) seem especially attractive because their amounts in the input feed can easily be increased. However, if sulfur or nitrogen compounds are

introduced through co-firing, it is important to obey good combustion practices; otherwise the increase in precursor concentrations can offset the inhibitory effects completely. In any case, it has been suggested that ammonia injection technology (AIT) can provide an effective means to the control PCDD/Fs (and acidic gases) in MWIs (32).

From a practical point of view, PCDD/F formation, and therefore inhibition, depends greatly on the characteristics of the incinerator, e.g., turbulence and residence time both in the furnace and in the postcombustion zone, so that no laboratory- or pilot-scale results are applicable as such to full-scale incineration plants. For example, recent findings suggest that lime and ammonia (cf. above) injections have only minor effects on dioxins in an industrial-scale fluidized bed incinerator (49). Accordingly, the trends noted here may not be directly generalized to full-scale plants. However, the results may provide low-cost dioxin preventative methods for small, minor, and hard-to-control sources. Finally, it should be emphasized that it is primarily kinetics not the thermodynamics that are responsible for the formation of PCDD/Fs and other toxic organochloro compounds in incinerators, although in the case of de novo formation there is some limited evidence to suggest that the isomer distribution for each dioxin homologue may be thermodynamically controlled (50, 51). In the future, as our knowledge of the reaction mechanisms and their activation energies increases, more detailed kinetic models should be developed in order to minimize PCDD/F emissions from incinerators. Since it seems that the particle concentration plays a significant role in the formation and inhibition mechanisms, the effect of particle size distribution and the surface area of the particles in particular should be studied in more detail.

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