# Inhibition of PCDD/PCDF formation during de novo synthesis on fly ash using N- and S-compounds.

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*Introduction*
Inhibition of formation of polychlorinated dibenzo-p-dioxins/dibenzofurans (PCDD/F) has been studied by various research groups. Basic substances (CaO, KOH, $Na_2CO_3$), elemental sulfur and functionalized amines all limit PCDD/F formation to some extent.[1,2,3] In this study we used EDTA, NTA, $Na_2S$ and $Na_2S_2O_3$ as inhibitors during PCDD/F formation from a carbon/fly ash mixture. These four compounds were chosen because of their ability to form stable complexes with transition metal ions (e.g. Cu) which act as a catalyst in fly ash. Formation of these complexes could reduce the catalytic activity of fly ash.

*Experimental*
From fly ash (MWI Zaanstad) all organic material was removed. 91% fly ash, 2% carbon (100-325 mesh), 5% NaCl and 2% inhibitor (EDTA, NTA, $Na_2S$, $Na_2S_2O_3$) were mixed by shaking. For experiments without inhibitor 93% fly ash, 2% carbon and 5% NaCl was used. 2.0 g of the mixture was placed in a cylindrical sample basket and coupled with a glass inlet tube for introduction of a gas flow through the fly ash bed. Sample basket and inlet tube were placed in a horizontal pyrex glass reactor, which had been pre-heated in a tube furnace for 30 min. All experiments were performed in duplo for 60 min at 348 °C (± 7 °C). Blank controls were made of fly ash, carbon, NaCl and all inhibitors. Starting materials contained between 1.6-8.7 ppb Σ PCDD/F. A gas stream (52 ± 3.5 ml/min $N_2$ and 6.5 ± 0.6 ml/min $O_2$) was passed through a washing-bottle containing water and then through the fly ash bed. The gas flow was controlled by Series 840 Side=Trak™ mass flow controllers. Nitrogen and oxygen were mixed in a mixing chamber (V=800 ml) containing ceramic pellets. Products evaporating from the fly ash surface were collected using a cold trap (80 ml toluene

cooled with ice). After the experiment the fly ash surface was extracted with a 3% HCl solution, dried overnight, spiked with 100 µl [13-C] PCDD/F mix as an internal standard for quantification, and soxhlet extracted with 400 ml of toluene (together with the toluene fraction from the cold trap) for 24 hrs. The extract of toluene was subjected to open column chromatography and preparative HPLC which separates the OCDF from the other PCDD/F. The sample was finally analysed by GC/MSD. $^{13}C_{12}$-1,2,3,7,8,9-H6CDD was used as a recovery standard. Average recoveries were (per isomer group): T4CDD 44%, T4CDF 45%, P5CDD 49%, P5CDF 56%, H6CDD 57%, H6CDF 53%, H7CDD 58%, H7CDF 57%, OCDD 56%.

*Results and discussion*

Table 1 shows results from experiments using the four different inhibitors. In all experiments a reduction of PCDD/F formation is observed. EDTA, NTA and $Na_2S$ give roughly the same reduction (75-80%), $Na_2S_2O_3$ is less good as an inhibitor (ca 30%).

The mechanism of inhibition is not well understood. Basic substances added to the fly ash could react with HCl and thus reduce its chlorinating ability. Interaction with the catalyst is not necessary. Complexation of the inhibitors used in this study with transition metal ions is likely but not proven. Recently, Lippert et al. have observed the existence of a Cu-N bond during the reaction of bromobenzene on alumina with Cu as a catalyst and ethanolamine as an inhibitor (200 °C).[4]

If only one route of formation with one catalyst exists during de novo synthesis, an inhibitor can only reduce the amount of PCDD/F formed but not change the [PCDD]: [PCDF] ratio, the congener or isomer distribution. Parallel formation pathways catalyzed by various species can be affected by an inhibitor in a different way and consequently such a change in ratio or pattern can occur.

In our experiments a change in the [PCDD]: [PCDF] ratio is seen with NTA. As can be seen in table 1, inhibition of PCDD formation is 92.3 ± 1.2% and 78.5 ± 1.3% for PCDF. If the ratio were to be the same as in the uninhibited experiment, these percentages should be equal. With the other three inhibitors, the change is small or not clear due to the margins of error.

A change in the congener distribution of PCDD and PCDF is seen with two inhibitors when comparing with the results of the uninhibited experiment. During this experiment - when setting ΣPCDD= 100% and ΣPCDF= 100% - formation of Σ T4CDD + P5CDD was 55.6 ± 3.1% and Σ T4CDF + P5CDF was 70.5 ± 1.1% . With EDTA Σ T4CDD + P5CDD= 73.0 ± 2.5 % and Σ T4CDF + P5CDF= 77.8 ± 3.2% ; with $Na_2S$ figures are 86.1 ± 3.5% and 90.3 ± 2.4% respectively. With these two inhibitors there is a tendency towards formation of lower chlorinated congeners. The other inhibitors - NTA and $Na_2S_2O_3$ - do not exhibit such a behaviour, percentages

Table 1, yields of PCDD/F ng/g fly ash. (a)

| | Σ PCDD + PCDF | % inhibition (b) | Σ PCDD | % inihibition (b) | Σ PCDF | % inhibition (b) |
|---|---|---|---|---|---|---|
| no inhibitor | 1900 ± 200 | | 226 ± 23 | | 1674 ± 172 | |
| EDTA | 460 ± 60 | 75.3 ± 4.7% | 34 ± 3 | 84.6 ± 2.8% | 422 ± 41 | 74.3 ± 5.1% |
| NTA | 370 ± 2 | 80.3 ± 2.2% | 17 ± 1 | 92.3 ± 1.2% | 356 ± 1 | 78.5 ± 2.3% |
| $Na_2S$ | 360 ± 10 | 80.7 ± 2.6% | 32 ± 1 | 85.6 ± 2.0% | 324 ± 10 | 80.4 ± 2.6% |
| $Na_2S_2O_3$ | 1330 ± 110 | 28.6 ± 13.3% | 187 ± 7 | 16.0 ± 11.7% | 1143 ± 103 | 30.3 ± 13.3% |

(a): All experiments 93% fly ash, 2% carbon, 5% NaCl or 91% fly ash, 2% carbon 2% inhibitor, 5% NaCl, $H_2O$ (g), 60 min at 348 ± 7 °C, 52 ± 3.5 ml/min $N_2$, 6.5 ± 0.6 ml/min $O_2$, in duplo, mean value ± range.

(b): Calculated relative to the experiments without inhibitor, e.g. for EDTA (460 ± 60) : (1900 ± 200) = 24.7 ± 4.7% formation is left, corresponding to 75.3 ± 4.7% inhibition.

of Σ T4CDD + P5CDD and Σ T4CDF + P5CDF formed are nearly equal to those in the uninhibited experiment.

The changes observed in either the [PCDD]: [PCDF] ratio (with NTA) or the congener distribution (with EDTA and $Na_2S$) lead to the conclusion that more than one formation route exists with different catalytic species acting to enhance rates of formation.

Figure 1 shows the various isomer distributions of T4CDD (Σ T4CDD= 100%) which are formed when using the four inhibitors. Little change is seen in the distribution, a result which has been encountered previously when varying such parameters as surface, reaction time, temperature or the presence of $H_2O$. The same trend is seen within the other isomer groups. Thus, inhibitors are capable of reducing the amounts of PCDD/F formed but not able to suppress formation of the toxic 2,3,7,8- substituted isomers selectively.

Further study is required for optimizing the concentrations of the inhibitor in order

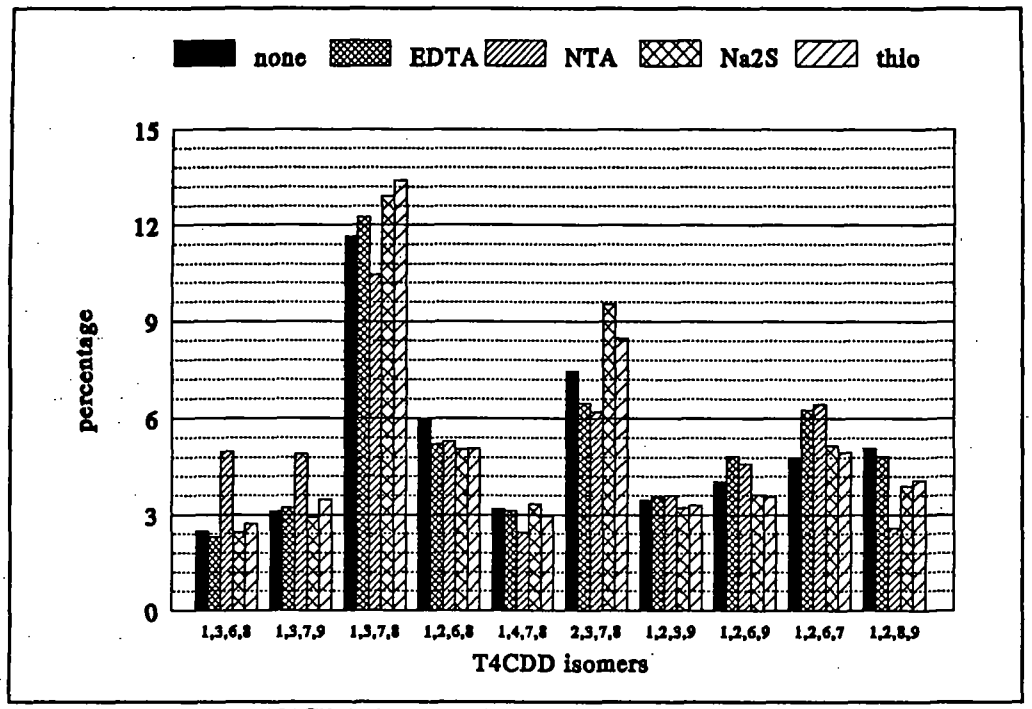


Figure 1 T4CDD isomer distribution with various inhibitors. (thio: $Na_2S_2O_3$)

to make reduction of formation as effective as possible.

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