Human fecal PCDD/F-excretion exceeds the dietary intake

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Summary

A mass balance of human dietary PCDD/F-intake and fecal PCDD/F-excretion was carried out. The participants of the study were seven male and seven female adults at the age of 24 to 64 years, living in North Rhine-Westphalia, Germany. The PCDD/F-intake was measured by the duplicate method. Sampling time of each food duplicate covered three days. The fecal PCDD/F-excretion was measured by collecting the feces corresponding to the food duplicates.

The mean daily dietary PCDD/F-intake was 49 pg I-TEq/d (range: 23 - 96 pg I-TEq/d) and therewith lower than estimated in the past. The mean daily fecal PCDD/F-excretion was 98 pg I-TEq/d (40 - 200 pg I-TEq/d). This is twice the amount of the PCDD/F-intake. Especially the fecal excretion of OCDD was higher than the dietary intake (mean: 7fold, range: 1.2 - 21fold).

The differences between PCDD/F-intake and PCDD/F-excretion may be caused by additional sources of exposure, *de novo* formation of PCDD/F in the human body or reduction of the body burden as a consequence of decreasing PCDD/F-intake.

Introduction

The estimation of the PCDD/F-exposure of men is usually performed by calculating the external (food, air etc.) and/or the internal (human blood, mother's milk etc.) exposure [1-5]. It turned out that this exposure decreased in the last years. Up to now the connection between the PCDD/F-intake by man and its elimination has only been studied with regard to nursed infants [6-9]. For adults however no data exist. In this study the relation between the dietary PCDD/F-intake and the fecal excretion was examined.

Materials and methods

The participants were 14 adults, seven male and seven female, at the age of 24 to 64 years, living in the Ruhr District in North Rhine-Westphalia, Germany. The samples were collected between December 1994 and February 1995. The dietary PCDD/F-intake was measured by the duplicate method. The sampling time for each food duplicate covered three consecutive days. The sampling days were regularly spread over workdays and weekends [10, 11].

For marking the stool specimen corresponding to the food duplicates the participants were asked to eat maize first at the morning of the first food-sampling day and last at the evening of the third food-sampling day. The participants were required not to eat maize both on the days before and after the food-sampling period and during the study.

The participants collected the stool specimen in precleaned aluminium bowles. To simplify the sampling the participants received a special compartment for the toilet in which they could place the bowles. After the defecation the bowles were closed with an aluminium top and stored in a plastic box. The bowles from the previous day and the glasses with the food duplicates were collected by us each day. The samples were weighed and frozen at -20°C. Feces sampling was stopped when the maize eaten on the evening of the third day had appeared in the feces. All stool specimen from one participant which contained at least 80 % of the marker from the first and the third day as well as the stool specimen from the intervening days were pooled and lyophilized. The dried sample was pulverized by pestle and mortar and the dry weight was measured. The feces samples were then stored at -20°C until analysis.

The food and the feces analyses were performed in series of 5 samples and 1 blank. About 60 g of the food lyophilisate or 15 g of the feces lyophilisate were spiked with 17 $^{13}C_{12}$-labelled PCDD/F-isomers and soxhlet-extracted with toluene:2-methoxyethanol (90+10) for 24 hours. The extract was evaporated at 40°C in a rotary evaporator until constant weight. The residue, which represents the fat content, was weighed, redissolved in hexane and cleaned-up with standard methods, including several types of modified silicagels, activated aluminia and activated charcoal. The purified extract was analyzed by HRGC/HRMS. Analytical conditions were described in detail elsewhere [12].

Results and discussion

In table 1 basic statistical data of food intake and fat consumption as well as the amount of stools and of fat excretion are shown.

Table 1: Mean values and ranges of food intake, fat consumption, amount of stools and of fat excretion of 14 adults

		Food intake (n=14)		Fecal excretion (n=14)	
		Mean	Range	Mean	Range
Dry weight	[g/d]	440	330 - 610	47	28 - 71
Fat weight	[g/d]	99	59 - 140	13	7.9 - 25

In all samples (food and feces) both 2,3,7,8-chlorosubstituted and non-2,3,7,8-chlorosubstituted PCDD/F were detected. In the feces the amount of non-2,3,7,8-chlorosubstituted PCDD/F as well as the sum of PCDF were considerably lower than in the food duplicates. The percentage of non-2,3,7,8-chlorosubstituted PCDD/F was only 3 % in the feces but 25 % in the food duplicates. The mean PCDF/PCDD-ratio was 35 % in the food duplicates and 8 % in the feces. This is mainly caused by higher OCDD-levels in the feces. The mean percentage of OCDD in the sum of PCDD/F was 76 % in the feces and only 43 % in the food duplicates. The PCDD/F-levels in the feces are comparable to those published by Rappe and Andersson (1992) [13].

Table 2 shows mean values and ranges of the dietary intake and the fecal excretion of selected 2,3,7,8-chlorosubstituted congeners and calculated TEq. As a rule the food and the feces - with regard to the calculated daily doses as well as to the concentrations of the 2,3,7,8-chlorosubstituted congeners - showed no correlation ($\alpha \leq 0.05$).

In figure 1 the dietary PCDD/F-intake is compared to the fecal PCDD/F-excretion on a dose basis. The surprising fact is that in the majority of the participants the daily fecal excretion of most of the 2,3,7,8-chlorosubstitued congeners exceeds their dietary intake. Figure 2 shows the calculated ratios of fecal PCDD/F-excretion to dietary PCDD/F-intake. A ratio larger than one indicates that the fecal excretion

exceeds the dietary intake. The higher excretion was observed mainly for OCDD (mean excretion was 7fold higher, range: 1.2fold - 21fold) followed by 1,2,3,6,7,8-HexaCDD (6fold higher) and 1,2,3,4,6,7,8-HeptaCDD (4fold higher). The mean excretion of OCDD exceeded the mean dietary intake by 2,300 pg/d in the average. The mean daily PCDD/F-excretion, calculated as I-TEq, is 2.4fold the mean dietary PCDD/F-intake (0.74fold - 5.3fold). Figure 3 presents the frequency distribution of the I-TEq on a dose basis.

Table 2: Dietary intake and fecal excretion of PCDD/F by adults (n=14) on a dose basis

	Dietary intake (n=14)		Fecal excretion (n=14)	
	Mean	Range	Mean	Range
	[pg/d]			
2,3,7,8-TetraCDD	6.6	3.3 - 14	11	3.1 - 20
OctaCDD	580	160 - 1300	2900	870 - 4600
2,3,4,7,8-PentaCDF	32	7.7 - 83	66	31 - 140
BGA/UBA-TEq	30	15 - 49	56	20 - 110
NATO/CCMS-TEq	49	23 - 96	98	40 - 200
	[pg/(kg$_{\text{body weight}}$ · d)]			
2,3,7,8-TetraCDD	0.098	0.026 - 0.26	0.15	0.048 - 0.27
OctaCDD	7.9	2.4 - 17	40	14 - 68
2,3,4,7,8-PentaCDF	0.48	0.059 - 1.5	0.92	0.48 - 1.7
BGA/UBA-TEq	0.44	0.18 - 0.89	0.78	0.32 - 1.3
NATO/CCMS-TEq	0.72	0.18 - 1.7	1.4	0.62 - 2.4

Figure 1: Mean values (+), medians (I), ranges (⊢, —⊣), 25. ([) and 75. (]) percentiles of the dietary PCDD/F-intake and the fecal PCDD/F-excretion on a dose basis [pg/(kg · d)] (only 2,3,7,8-chlorosubstituted congeners or the sum of them are presented)

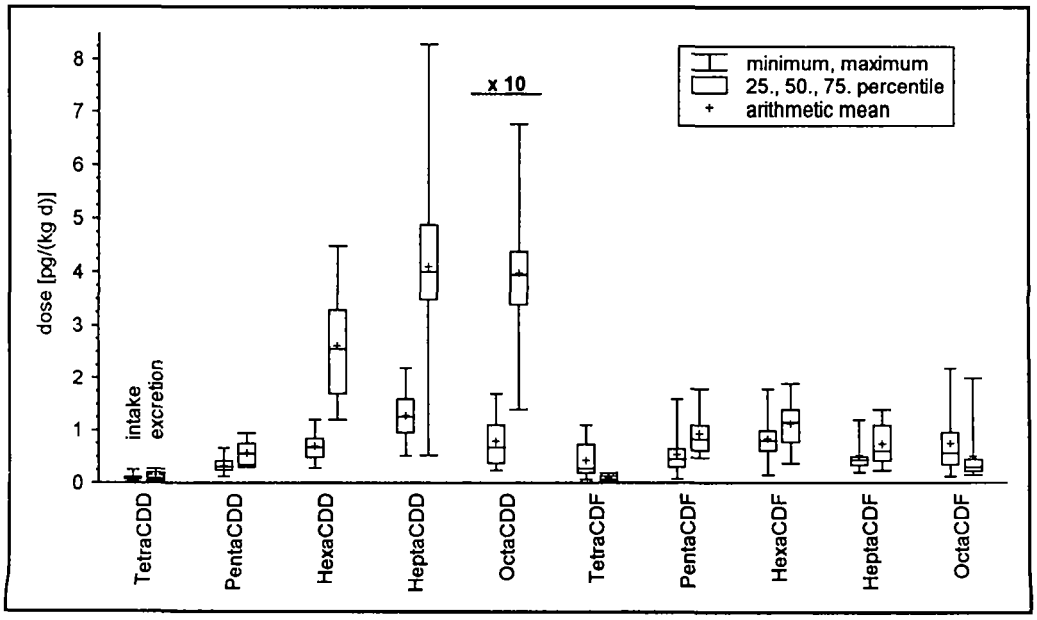


Figure 2: Ratio of fecal PCDD/F-excretion to dietary PCDD/F-intake of 14 adults

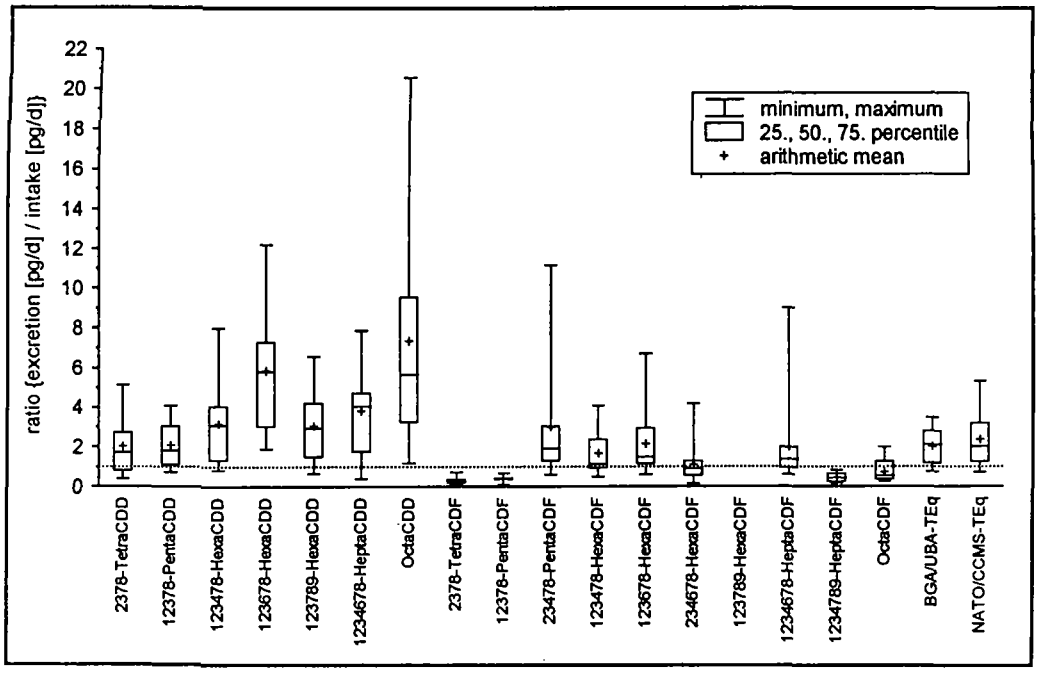
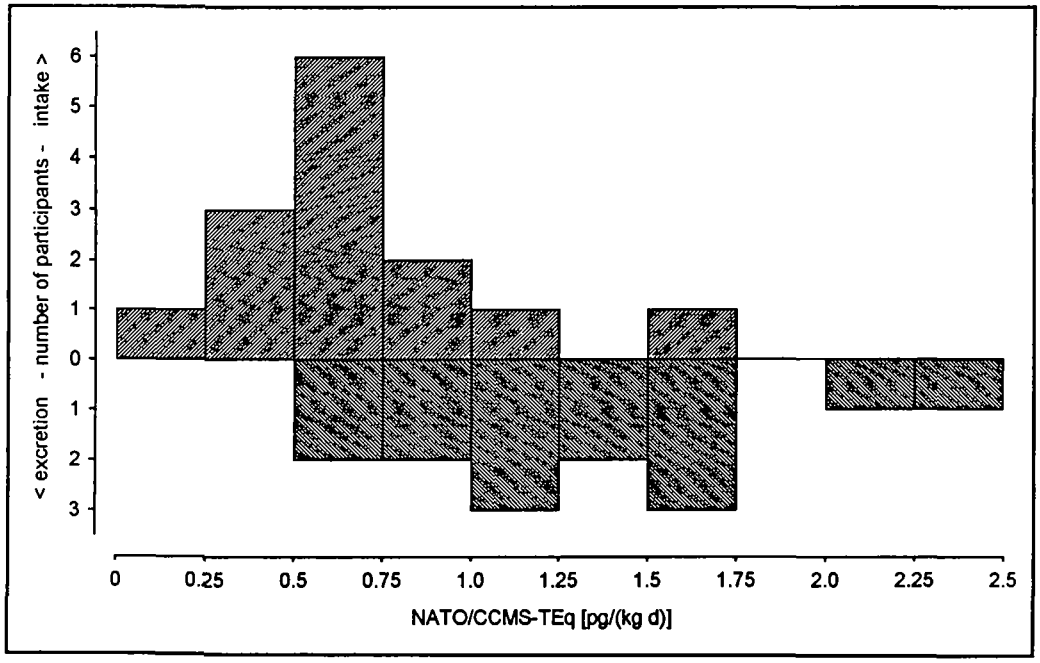


Figure 3: Dietary PCDD/F-intake and fecal PCDD/F-excretion, calculated as I-TEq on a dose basis [pg/(kg · d)]



The differences between PCDD/F-intake and excretion could be explained by several ways:

- Missing sources of exposure
  It has been assumed that more than 90 % of the PCDD/F-intake takes place via food whereas other sources such as inhalation of air or ingestion of soil are of minor importance. Especially the high levels of OCDD in feces couldn't be caused by the actual OCDD-levels in food. This was also assumed by Rappe and Andersson [13] who measured high levels of OCDD in feces, too.
  To calculate a contribution of other sources to the daily intake the PCDD/F-levels in house dust [14-16] and on human skin [17, 18] were determined. Although it was not possible to calculate exactly the intake of house dust and the resorption of PCDD/F by human skin these additional sources can not explain the whole difference between intake and excretion of PCDD/F. Other sources of exposure may be presently unknown.

- *De novo* formation of PCDD/F in men
  In compost a formation of PCDD/F, especially Hepta- and OctaCDD, has been observed. It was assumed that for this formation the presence of precursers for example chlorophenols, is necessary [19].
  In the presence of peroxidases and hydrogen peroxide a transformation of Pentachlorophenol (PCP) to OCDD was found in vitro. 0.1 mg OCDD was formed per 1 g PCP [20] (0.01 %).
  In the gastro-intestinal tract of men however, a formation of OCDD or other PCDD/F is presently unknown. Since the presence of precursers such as PCP in food and human blood is well known, the possibility of the *de novo* formation of PCDD/F should be taken into account. For example the PCP-levels in human blood are in the range of some µg/l, and therewith about 10^6fold higher than the PCDD/F levels in blood. With regard to the body burden of PCP a formation of OCDD in the concentration range as measured in the feces seems to be possible. This problem is presently under investigation in our laboratories.

- Reduction of the body burden as a consequence of decreasing dietary intake
  The measures taken to reduce the PCDD/F-emissions led to lower PCDD/F-levels in the environment. As a consequence we have shown that the dietary PCDD/F-intake by adults is lower than estimated in the past [10]. Additionally within the last years a decrease of the PCDD/F-levels in samples of human origin, for example mothers's milk and human blood, has been observed. The PCDD/F-concentrations measured in the blood of the participants of this study were also lower than the blood-levels of unexposed persons of former studies. Therefore it can be suspected that the high PCDD/F-excretion - in relation to the low actual dietary intake - is a result of the reduction of the body burden.

Conclusions

The mean daily dietary PCDD/F-intake is lower than estimated in the past. The majority of the participants show - with regard to most of the 2,3,7,8-chlorosubstituted congeners - a higher fecal PCDD/F-excretion than a dietary intake of these substances. This is on the average twice for the calculated I-TEq and 7fold for OCDD respectively.
The differences may be due to additional sources of exposure, *de novo* formation of PCDD/F in man or reduction of the body burden as a consequence of decreasing PCDD/F-intake. Further investigations are necessary to give answers to this open questions.

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