PCDD/PCDF in Humans - An Update of Background Data

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ABSTRACT

44 blood samples - collected in 1992 in Germany - have been analysed for PCDD/PCDF. For all persons involved, no certain expose, except for food consumption, has been reported. The levels measured show a decrease of PCDD/PCDF compared to previous data on blood collected mainly in 1989. This trend for the Dioxins values in blood corresponds well to the observations reported for human milk.

INTRODUCTION

Fürst et al.[1] and Beck et al.[2] reported the first german background data on PCDD/PCDF in human milk as well as in human adipose. The corresponding blood data were reported by us in 1989 - starting with 10 donors[3] with no known exposure and extending the reference group to 102 persons later on[4]. In 1992 we observed that in an increasing number of cases the levels in patients without known exposure were lower than the reference data from 1989.

MATERIALS AND METHODS

The whole blood samples were frozen immediatley after collection, transported to the laboratory and kept frozen until analysed for PCDD/PCDF. For all 44 blood samples the inquieries resulted in no indication for any other exposure as food consumption. The analytical method used were nearly identical to those applied for the successful participation in the WHO interlaboratory validation studies (round II and III) on human blood and will not be described here[5,6,7].

RESULTS AND DISCUSSION

The statistical data from this investigation is presented in Table 1. The TEQ values have been calculated according to the NATO-CCMS (I-TEQ) as well as to the German Federal Health Office (FHO) models.

Values in pg/g (ppt), lipid based

ISOMER	MIN	MAX	MEAN	STD	MEDIAN
Age	23	63	36.8	11.64	31
2.3.7.8-Tetra-CDD	1.0	8.8	3.7	1.75	3.3
1.2.3.7.8-Penta-CDD	2.8	20.8	8.3	3.65	7.7
1.2.3.4.7.8-Hexa-CDD	3.6	19.4	10.2	4.55	9.0
1.2.3.6.7.8-Hexa-CDD	7.5	99.0	35.5	17.59	30.7
1.2.3.7.8.9-Hexa-CDD	1.8	15.8	5.9	2.45	5.6
Total Hexa-CDD	13.9	125.0	51.9	22.34	46.8
1.2.3.4.6.7.8-Hepta-CDD	16.7	159	56.7	30.87	48.6
Octa-CDD	123	1267	462	224.67	418
2.3.7.8-Tetra-CDF	1.2	3.8	2.1	0.67	2.1
1.2.3.7.8-Penta-CDF	n.d.	2.5	0.4	0.74	n.d.
2.3.4.7.8-Penta-CDF	6.8	48.2	18.8	10.47	16.3
Total Penta-CDF	6.8	50.2	19.2	10.68	17.5
1.2.3.4.7.8-Hexa-CDF	4.4	24.5	10.9	4.85	9.7
1.2.3.6.7.8-Hexa-CDF	3.1	20.7	7.8	4.06	7.0
1.2.3.7.8.9-Hexa-CDF	n.d.	1.2	n.d.	0.18	n.d.
2.3.4.6.7.8-Hexa-CDF	n.d.	9.9	2.9	2.25	2.4
Total Hexa-CDF	7.9	49.4	21.6	10.32	19.8
1.2.3.4.6.7.8-Hepta-CDF	8.5	38.4	19.0	6.20	17.6
1.2.3.4.7.8.9-Hepta-CDF	n.d.	2.4	0.4	0.74	n.d.
Total Hepta-CDF	8.5	38.4	19.4	6.16	18.1
Octa-CDF	n.d.	14.8	4.0	3.12	3.1
Total PCDD	182.1	1526.6	581.7	260.34	547.4
Total PCDF	32.2	141.5	66.3	24.76	65.8
Total PCDD/PCDF	235.0	1634.6	648.0	271.91	607.8
I-TEQ (NATO-CCMS)	**12.0**	**61.0**	**26.0**	**11.06**	**24.1**
TEQ (BGA/UBA)	**7.4**	**33.5**	**15.2**	**5.82**	**14.4**

n.d. = not detectable

Table 1. PCDD/PCDF in whole blood; background, 1992, n = 44

Comparing the data of the 1992 blood to values from blood collected between 1988 and 1990, mainly 1989, it is obvious, that nearly for all isomeres the mean, median and maximum levels are lower in the 1992 group (for comparison / I-TEQ / 1989: mean = 40.8 pg/g, median = 37.8 pg/g, maximum = 93.5 pg/g).

This so called 'time trend' has been observed by other authors as well. Fürst et al.[9] and Norén et al.[11] analysed human milk for all PCDD/F-isomeres while Patterson[12] reported only the decrease of 2.3.7.8-TCDD in blood.

In Figure 1 our new blood results - expressed in I-TEQ values - are compared to those for adipose, human milk and blood from other authors. It must be taken into account, however, that the mean age varies from group to group.

Author	Matrix	n	Ref.	Age, mean
Beck et al.	Adipose	20	2	50
Päpke et al.	Blood	102	4	37
Kieselrotstudie	Blood	56	8	41
Päpke et al.	Blood	44	This paper	37
Fürst et al.	Human milk	555	9,10	~ 30

It is striking that a distinct decline of the I-TEQ values with time can be observed (Fig. 1).

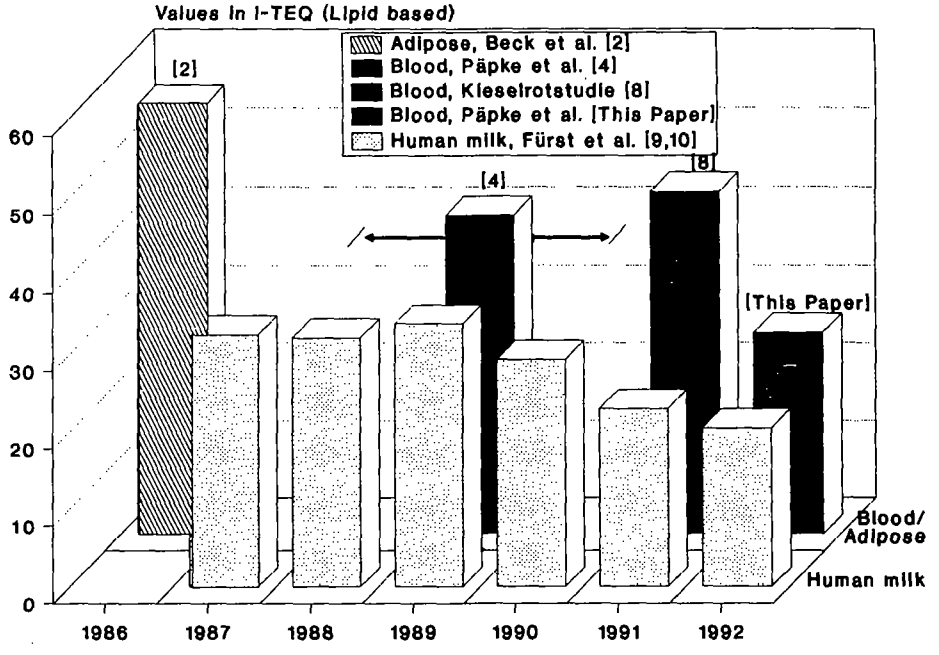

Figure 1 Time trend of PCDD/F, 1986-1992, Means

It can not be excluded that the observed effects results from reducing the PCDD/-PCDF emissions from such sources as municipal waste incinerators, cars (unleaded gasoline) as well as the ban on use and production of PCP and in the pulp bleaching technologies.

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