LEVELS OF 2,3,7,8-SUBSTITUTED POLYCHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS (PCDD/PCDF) IN HUMAN LIVER AND ADIPOSE TISSUE

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ABSTRACT

Liver and adipose tissue samples of 21 Swiss inhabitants of known sex, age, body weight, stature and cause of death were analyzed for toxic PCDD/PCDF. Analysis by HRGC/MS was performed subsequent to a clean-up according to the procedure described by Smith et al. [1] which had been slightly modified and automated. In liver (and adipose tissue, data in brackets) the average concentrations were: 12378-PeCDD: 1.1 (24.1) ppt, 123478-HxCDD: 1.1 (15.3) ppt, 123678-HxCDD: 10.5 (144.3) ppt, 1234678-HpCDD: 81 (195.1) ppt, OCDD: 490.5 (1161) ppt, 2378-TCDF: 0.2 (0.8) ppt, 12378-PeCDF: 2.3 (3.3) ppt, 23478-PeCDF: 7.3 (48.5), 123478-HxCDF: 3.7 (8.3) ppt, 123678-HxCDF: 4.7 (7.4) ppt, 234678-HxCDF: 1.8 (8.0) ppt, 1234678-HpCDF: 1.6 (1.2) ppt, OCDF: 0.2 (0.4) ppt.
No age or sex related trends in tissue levels could be observed.
Distribution of the congeners between the two tissues was different from that observed in the rat nor was a similar congener-specificity exhibited.

EXPERIMENTAL

Extraction and clean-up of the samples was performed according to the method of Smith et al. [1] which was partly modified and automated, using a Kontron Tracer-670 valve switching unit for the first part of the enrichment procedure (Fig. 1). Recovery of the whole clean-up procedure, tested with ^{14}C-TCDD, was 75.8 %. HRGC/MS was used for identification and quantification (20 m glass capillary, coated with OV-240-OH [2], carrier gas: H_2, MS Finnigan MAT 4510 operated in the multiple ion monitoring mode, using negative chemical ionization), referring to ^{13}C-labeled internal standards which had been added to the samples prior to clean up.

Human liver and adipose tissue samples were obtained from the pathology department of the University Hospital of Zurich. The age of the individuals ranged from 15 to 85 years (average 47 years). Further information, i.e. sex, body weight, stature, profession and cause of death were also accessible.

RESULTS

Fig. 2 show the individual data points (and mean) of PCDD/PCDF levels detected in adipose and liver tissues, respectively. Levels of 2378-TCDD were below the detection limit. PCDD concentration in both compartments increased with increasing degree of chlorination, with OCDD being the dominating isomer. Of the PCDFs, 23478-PeCDF and the hexachlorinated isomers made the largest contribution to the body burden. Values are similar to those reported by Thoma et al. [3] for people from the Munich area.

Means of the individual adipose to liver tissue concentration ratios of the different congeners are shown in Table I. With one exception (1234678-HpCDF), levels in the adipose tissue were higher than in the liver. Among the congeners with the same degree of chlorination, adipose/liver concentration ratios were higher for the PCDDs. The ratios did not differ much among the two HxCDDs and the three HpCDFs. For 23478-PeCDF this ratio was about 4 times higher than for 12378-PeCDF.

DISCUSSION AND CONCLUSIONS

In contrast to the rat, where liver contains most of the toxic PCDD/PCDF body burden, body fat is the major storage site of PCDD/PCDF in humans. No parallel between the two species was noted in the congener-specific body distribution. For example, the different disposition of the two toxic PeCDFs in rats (liver-to-adipose concentration ratio of 12378-PeCDF: 4-5, 23478-PeCDF: 40-45 [4]) was not apparent in humans, where even the 23478-PeCDF seems to be retained preferably in the body fat.

Thus, an assessment of a toxic risk for humans on basis of rat toxicity data, would require the inclusion and consideration of the different kinetics and target tissue levels of the PCDD/PCDF.

No correlation was found between age or weight (body fat content) and tissue levels (data not shown). However, an establishment of such correlations would require a larger group of samples and also some knowledge of the exposition level of the individuals.

References

[1] L.M. Smith et al. (1984); Anal. Chem., 58, 1830-1842
[2] P. Schmid (1989); J. High Res. Chrom., 12, 665-668
[3] H. Thoma et al. (1990); Chemosphere, 20, 433-442
[4] K. Abraham et al. (1989); Arch. Toxicol., 6, 193-202

Fig. 1: Scheme of the first part of enrichment procedure for PCDD/PCDF.

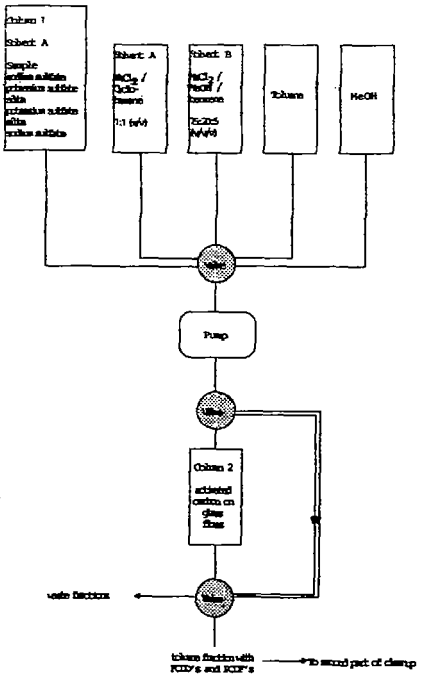

Table 1: Adipose-/ Liver tissue concentration ratios

Congener	Adipose/ Liver	S.D.
2,3,7,8 -TCDF	4.6	0.8
1,2,3,7,8 - PeCDD	21.1	6.6
1,2,3,7,8 - PeCDF	1.4	0.8
2,3,4,7,8 - PeCDF	6.6	2.6
1,2,3,4,7,8 - HxCDD	14.6	6.8
1,2,3,6,7,8 - HxCDD	13.8	6.1
1,2,3,4,7,8 - HxCDF	2.2	0.9
1,2,3,6,7,8 - HxCDF	1.6	0.6
2,3,4,6,7,8 - HxCDF	4.4	2.6
1,2,3,4,6,7,8 - HpCDF	0.7	0.6
1,2,3,4,6,7,8 - HpCDD	2.4	0.9
OCDF	2.1	1.3
OCDD	2.4	0.8

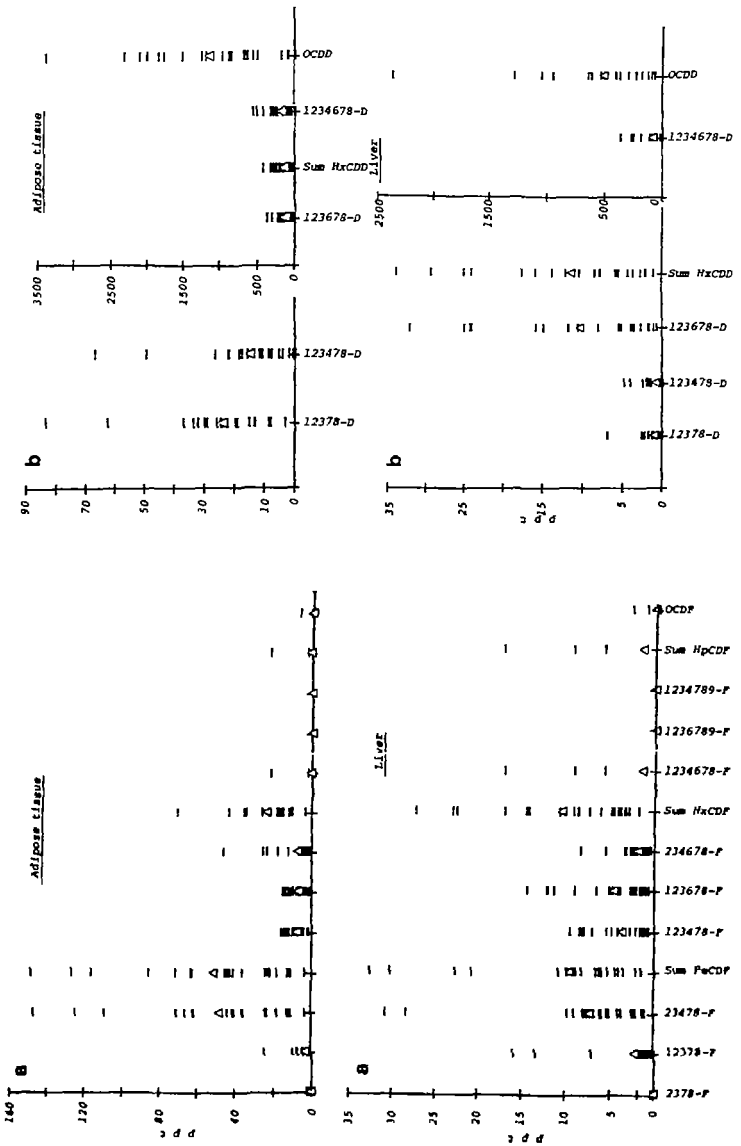

Figure 2: PCDF (a) and PCDD (b) in human liver and adipose tissue (ppt on wet weight basis). Individual data and mean (n=21).