Time trends in human dietary exposure to PCDDs, PCDFs and PCBs in the UK

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1. Introduction

It is generally accepted that dietary intakes of PCDDs, PCDFs and PCBs account for most of human exposure to these compounds, in the absence of occupational or accidental exposure. Previous surveys have been reported of PCDDs and PCDFs [1] and PCBs [2] in a range of UK foods. For PCDDs and PCDFs, results were reported for a relatively small number of samples. Data on PCBs in the UK diet were limited to total PCBs measured as Aroclor equivalents.

To provide more reliable estimates of recent average UK dietary intakes and to investigate trends over the last decade, congener specific analysis of PCDDs, PCDFs and a large number of PCBs has been undertaken on samples of fatty foods from the UK Total Diet Study (TDS) collected in 1982 and 1992.

The TDS has been described in detail elsewhere [3]. Retail samples of 115 specified food items are purchased at 2 week intervals from different locations in the UK, prepared as for consumption, then combined into one of 20 food groups in proportions representing the relative importance of the retail foods in the average UK diet. When all or most of the sample sets collected in a year are analysed, the use of TDS samples ensures a large, diverse base of individual samples sourced from across the UK and composite samples representative of national dietary habits.

It is also well established that these compounds accumulate in fatty tissues and are excreted in human breast milk resulting in exposure of the nursing infant. Previously, as part of a study organised by the World Health Organisation Regional Office for Europe (WHO/EURO) [4], the concentrations of PCDDs and PCDFs were determined in pooled samples of human milk collected in 1987/88 [5]. Further samples were collected to a comparable protocol in 1993/94 and their analysis is reported.

2. Methods

Concentrations of PCDDs, PCDFs and PCBs were determined in archived TDS samples of fatty foods and bread collected in 1982 and 1992. For each food group, the material analysed was a composite of samples from all 24 locations included in the TDS in that year. The contribution of fruit, vegetables and other non-fatty foods to total dietary intakes of PCDDs, PCDFs and PCBs is low [6]. These food groups were not analysed in the current survey.

PCDDs and PCDFs were also determined in three composite samples of human milk, one from each of Birmingham, Glasgow and Cambridge. Each sample was a composite of milk collected in 1993-94 from at least twenty donors.

Food and human milk samples were freeze-dried, spiked with $^{13}C_{12}$-labelled internal standard then extracted with dichloromethane/cyclohexane. The extract was passed through beds of silica gel and base-modified silica, and fractionated using a column containing activated carbon dispersed on glass fibres. Details of the fractionation procedure have been published [7]. Each fraction was then purified by chromatography on acid-modified and base-modified silica.

PCDDs, PCDFs and non-*ortho* PCBs were determined in the appropriate fractions by high resolution gas chromatography/mass spectrometry (GC/MS) at 7000 resolution. Trichlorinated PCBs in the fraction containing *ortho*-chlorinated PCBs were also determined by high resolution GC/MS. Other PCBs were determined in the appropriate fraction by low resolution GC/MS. Reporting limits were 0.01 ng/kg whole product for PCDDs, PCDFs and non-*ortho*-PCBs, and 5.0 ng/kg whole product for other PCBs. All data for PCDDs and PCDFs met published acceptance criteria [8]; data for PCBs met comparable criteria.

The following PCB congeners were determined in TDS samples: PCBs 18, 28, 31, 33, 37, 41, 44, 47, 49, 51, 52, 60, 61, 66, 74, 77, 80, 81, 87, 99, 101, 105, 110, 114, 118, 123, 126, 128, 129, 138, 141, 149, 151, 153, 156, 157, 167, 169, 170, 180, 183, 185, 187, 189, 191, 193, 194, 201, 202, 203, 206, 208 and 209

3. Results

The concentrations of PCDDs and PCDFs and of PCBs in TDS samples are shown in Table 1. Totals represent the *upper bound* total concentrations. For each congener present at less than the reporting limit, this assumes that the concentration of that congener is equal to its reporting limit. PCDDs, PCDFs and PCBs were found in highest concentrations in fatty foods, whilst in bread most congeners were present below the limits of detection (LOD).

The concentrations of 2,3,7,8-chlorinated PCDDs and PCDFs in each sample of human milk are shown in Table 2. Data are also included for samples collected in 1987-88 and analysed as part of earlier surveillance [5]. The concentrations in the current survey were in the range 21 to 24 ng TEQ/kg on a fat basis, corresponding to 0.64 to 0.75 ng TEQ/kg on a whole milk basis.

4. Discussion and conclusions

Dietary intakes of PCDDs, PCDFs and PCBs by adults

In the UK, the International Toxic Equivalency Factor (I-TEF) system is used by regulatory agencies in the risk assessment of mixtures of PCDDs and PCDFs, but TEFs proposed for PCBs are not in use. A Tolerable Daily Intake (TDI) of 10 pg/kg body weight/day for 2,3,7,8-TCDD was recommended by an expert group convened by the WHO/EURO in 1990 [9]. The UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) endorsed this TDI in 1992 and 1995, recommending that it could be regarded as equivalent to an intake of 600 pg TEQ/day of PCDDs and PCDFs for a 60 kg adult [1,10].

The dietary intakes of PCDDs, PCDFs and PCBs have been estimated for average and high level (97.5th percentile) adult UK consumers by combining data from the analysis of TDS samples with food consumption data from the dietary and nutritional survey of British adults. In this study all food

eaten in a 7 day period by each of over 2000 adults was recorded. Estimates of the intakes from food groups which were not analysed have been included by assuming that each congener was present at its reporting limit.

Using this method, the estimated mean UK dietary intake of PCDDs and PCDFs has fallen from 250 pg TEQ/person/day in 1982 to 88 pg TEQ/person/day in 1992. High level intakes of PCDDs and PCDFs have also decreased considerably, from 442 pg TEQ/person/day in 1982 to 156 pg TEQ/person/day in 1992. Mean UK dietary intakes of the sum of the 53 PCB congeners determined have also fallen, from 1.0 µg/person/day in 1982 to 0.34 µg/person/day in 1992. High level intakes of these PCBs have fallen from 1.9 µg/person/day in 1982 to 0.60 µg/person/day in 1992.

Average and high level UK dietary intakes of PCDDs and PCDFs between 1982 and 1992 were considerably below the TDI endorsed by the COT. The estimated average UK dietary intake of dioxins in 1992 is similar to recent estimates in other industrialised nations. Average and high level UK dietary intakes of PCBs are considerably lower than the estimate of average UK dietary intakes of total PCBs made in 1983 of not greater than 40 µg/person/day and probably less than 10 µg/person/day [2]. The COT is reviewing the considerable body of recent research on the toxicity of individual PCB congeners and mixtures of PCBs.

A number of different schemes have been proposed to assign TEFs to PCB congeners. The validity of using TEFs for PCB congeners with suggested 'dioxin-like' Ah-receptor activity is being considered by the COT. The formal interpretation of the results of this survey has not included any assessment of the total TEQ of the PCBs measured in food, pending the outcome of the COT review. If, however, the TEQ system proposed by the 1993 WHO/IPCS consultation [1] were applied to concentrations found in this survey, the total average UK dietary intakes of PCDDs, PCDFs and PCBs would have fallen from 400 pg TEQ/person/day in 1982 to 140 pg TEQ/person/day in 1992.

There was a decrease between 1982 and 1992 in the concentrations of the majority of PCDD, PCDF and PCB congeners quantified in the majority of composite food group samples. There were small apparent increases between 1982 and 1992 in concentrations of PCDDs and PCDFs on a TEQ basis in bread and cereals, and in concentrations of the sum of 53 PCB congeners in bread and milk. This may have resulted from the use of *upper bound* estimates, which may significantly overestimate total concentrations in samples where most congeners are not present at these limits.

The composition of some of the food group samples will have been changed between 1982 and 1992 to reflect changes in household consumption. The fall in the fat content of the UK diet will have contributed to the observed decreases in dietary intakes of PCDDs, PCDFs and PCBs. Other changes in dietary habits, for example the increase in the proportion of dietary fat that is of vegetable rather than animal origin, may also have contributed to the observed decrease in dietary intakes of these contaminants.

Dietary intakes of PCDDs and PCDFs by breast-fed infants

A study which tracked the consumption of human milk by 48 infants was used to give estimates of the mean consumption of human milk by nursing infants between 2 and 10 months of age [12]. Multiplying these consumption figures by the average levels of PCDDs and PCDFs in human milk samples collected in 1993-94 gives estimates of mean intakes of these compounds from human milk. Estimated intakes from human milk are 110 pg TEQ/kg bodyweight/day at age 2 months, falling to

26 pg TEQ/kg bodyweight/day at 10 months of age due to an increased bodyweight and the move to a mixed diet.

These estimates of PCDD and PCDF intake may over-estimate the actual dietary exposure to these compounds from human milk for two reasons. First, concentrations of PCDDs and PCDFs in the mothers' milk will decrease over the period of breastfeeding by approximately 12% per month [13]. As the analysed samples of human milk met the WHO/EURO criterion of being collected from mothers no more than 2 months after delivery, these data may significantly over-estimate the actual intakes of PCDDs and PCDFs from human milk by nursing infants older than 2 months. Second, all samples were obtained from mothers nursing their first child. Levels of PCDDs and PCDFs in breast milk from mothers nursing their second child are 20-30% lower [14].

The concentrations of PCDDs and PCDFs in the human milk samples obtained in 1993-94 were approximately 35% lower than concentrations in corresponding samples collected in 1987-88. These findings are consistent with the reported decreases in levels of PCDDs and PCDFs in human milk in over the same period in other European countries.

The estimated dietary intakes of PCDDs and PCDFs by breast fed infants found in the current survey exceed the TDI. The COT has considered at length the implications of this erosion of the safety margin inherent in the TDI. The period of breast feeding is short compared with the long biological half-lives of 2,3,7,8-TCDD and related compounds in humans [15]. In consequence there would be insufficient time for the body burden in babies to rise to a high level. Taken over a lifetime, the eventual body burden would not be greatly increased by a short period of higher intake. Breast feeding confers considerable benefits on the young infant in terms of immunological protection, nutrition and mother-infant bonding. In its 1995 statement, the COT reiterated its support of the recommendation made by the WHO Working Group which considered this issue in 1988 [16]. The Working Group recommended that, despite the presence of dioxins in human milk, breast feeding should be encouraged and promoted on the basis of convincing evidence of its benefits to the overall health and development of the infant.

5. References

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Table 1. Concentrations of PCDDs, PCDFs (ng TEQ/kg whole food) and PCBs (µg/kg whole food in Total Diet Study samples collected in 1982 and 1992

Food group	PCDD/Fs		Sum of 53 PCB congeners	
	1982	1992	1982	1992
Bread	0.02	0.03	0.29	0.29
Other cereals	0.13	0.17	2.7	0.52
Carcass meat	0.49	0.13	1.1	0.36
Offals	1.6	0.59	0.56	0.33
Meat products	0.32	0.08	1.5	0.63
Poultry	0.50	0.13	1.8	0.59
Fish	0.41	0.21	3.8	1.1
Oils and fats	1.3	0.20	8.4	0.85
Eggs	0.92	0.17	2.4	0.52
Milk	0.16	0.06	0.35	0.37
Milk products	1.2	0.16	2.2	0.47

Table 2. Concentrations of PCDD/Fs (ng/kg fat basis) in composite samples of human milk from UK locations

	1987-88		1993-94		
Congener	Birmingham	Glasgow	Birmingham	Glasgow	Cambridge
2,3,7,8-TCDD	6.5	4.6	3.5	3.1	3.7
1,2,3,7,8-PeCDD	14	12	9.0	8.6	9.9
1,2,3,4,7,8-HxCDD	*	*	9.2	9.6	11
1,2,3,6,7,8-HxCDD	67*	57*	27	27	32
1,2,3,7,8,9-HxCDD	10	6.5	6.5	6.4	7.5
1,2,3,4,6,7,8-HpCDD	76	65	31	40	47
OCDD	303	271	130	170	190
2,3,7,8-TCDF	1.4	0.9	1.0	0.78	0.82
1,2,3,7,8-PeCDF	0.5	0.3	0.29	0.30	0.47
2,3,4,7,8-PeCDF	25	19	14	15	16
1,2,3,4,7,8-HxCDF	8.3	7.2	4.2	4.2	4.4
1,2,3,6,7,8-HxCDF	7.8	5.0	3.6	3.6	4.0
1,2,3,7,8,9-HxCDF	ND	ND	0.07	<0.06	0.09
2,3,4,6,7,8-HxCDF	3.6	2.3	2.0	2.2	2.5
1,2,3,4,6,7,8-HpCDF	9.5	7.1	2.9	4.0	4.0
1,2,3,4,7,8,9-HpCDF	ND	ND	0.13	0.15	0.19
OCDF	6.8	6.9	0.72	0.81	0.57
Total (ng TEQ/kg fat)	37	29	21	21	24
Fat content (%)	2.8	3.4	3.1	3.4	3.2

* = 1,2,3,4,7,8-HxCDD and 1,2,3,6,7,8-HxCDD could not be resolved and the figure given is for the sum of these two congeners.

ND = not detected