Human Exposure P1

Dioxin-like PCBs in food- their significance to human TEQ exposure

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Introduction

The UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) has recently recommended the use of Toxicity Equivalent Factors (TEFs) to assess the potential toxicity of complex mixtures of dioxin *and* selected polychlorinated biphenyls (PCBs) congeners present in food (COT, 1997). In this way, a Tolerable Daily Intake (TDI) of 10 pg TEQ/kg bw/day can be used to assess the health risks of intake of mixtures of PCDD/F and PCB congeners. The significance of including dioxin-like PCBs in the human risk assessment process in comparison to using a TEQ based on only PCDD/Fs is considered. An extensive literature search has been conducted and the extent of dioxin-like PCB contamination in a variety of foods evaluated. Also, the significance of background exposure levels and body burdens of PCBs is compared to that of PCDD/Fs.

Relative importance of PCB and PCDD/F TEQs in food

The TEQ composition of principal foods has been evaluated. Where possible, this has only included studies where <u>both</u> dioxin-like PCBs and PCDD/Fs have been analyzed. WHO/IPCS TEFs were used to calculate TEQs for the dioxin-like PCBs. For many food stuffs listed in Table 1, dioxin-like PCBs dominate Σ TEQ. This is particularly noticeable for the fish food group in which Σ TEQ is dominated by dioxin-like PCBs (up to 90% of Σ TEQ) - these data highlight the significance of fish consumption to human TEQ exposure. Additionally, in this respect, a recent UK survey (MAFF 1997b) has demonstrated increased exposure to PCB-TEQ as a result of ingesting fish oil supplements, the TEQs of which are dominated by the dioxin-like PCBs (from between ~60 to 90% of Σ TEQ). In summary, dioxin-like PCBs associated with animal fats and - more notably - fish represent a significant source of TEQ.

One of the most comprehensive dietary exposure studies available in terms of both food composition and temporal exposure information is the UK Total Diet Study. UK Total Diet Study (TDS) samples collected in 1982 and 1992 demonstrate that average dietary intake of PCBs has declined from 1.0 ug/person/day in 1982 to 0.34 ug/person/day in 1992 (MAFF 1997a). In 1982, estimated upper bound Σ TEQ intake in the UK was 4.1 pg TEQ/ kg body weight/day, of which 2.7 pg TEQ/kg body weight/day (~40%) came from PCBs. In 1992, intake had declined to 1.5 pg TEQ/kg body weight/day and 0.9 pg TEQ/kg body weight/day (~38%) respectively. Despite this decline in Σ TEQ intake, the proportion of TEQ from dioxin-like PCBs has, for certain major food groups (meat, oils and fats, dairy produce and eggs) increased from

ORGANOHALOGEN COMPOUNDS Vol. 38 (1998) 1982 to 1992. This is particularly noticeable for cows milk sampled in 1982, 1992 and 1995, in which the proportion of TEQ from PCBs has increased from 38% in 1982 to 64% in 1995 (MAFF 1997c).

Table 1 PCB and PCDD/F concentrations (expressed as ΣTEQ in ng/kg lipid) for a variety of fish reported in the literature, together with the percentage contribution of ΣTEQ attributable to PCB.

Media	PCB-TEQ	PCDD/F-TEQ	%PCB TEQ	Reference				
Fish and fish products (ng TEQ/kg fat)								
Eels (Germany)	75-120	12-23	82-85	Malisch 1996				
Eels (Germany)	19-288	82-119	32-71	Behnisch 1997				
Eels (UK)	8-87	2.6-15	73-92	MAFF 1997a				
Eels (New Zealand) n= 16 (pg/g wet wt)	0.23	0.028	89	Buckland et al., 1997				
Arctic Char (Finland) pg/g wet wt.	0.05-0.2	0.06-0.1	46-70	Vartiainen et al., 1996				
Trout (New Zealand) n=11 (pg/g wet wt)	0.057	0.018	76	Buckland et al., 1997				
Salmon (Sweden)	123	56	70	Ahlborg et al., 1994				
Seafish (Japan)	9.4	0.87	92	Masuda et al., 1996				
Market fish (Japan) (ng TEQ kg wet)	0.22	0.33	40	Masuda et al., 1996				
Fish (f.water) Toronto(ng TEQ kg wet)	0.36	0.26	58	Ryan et al., 1997				
Fish (freshwater) Montreal	0.32	0.16	67	Ryan et al., 1997				
Fish (marine) Toronto	0.24	0.033	86	Ryan et al., 1997				
Cod Liver oil bottled 1996	24-31	6.2-9.2	77-80	MAFF 1997b				
(n=8) ng TEQ/kg oil								
Cod liver oil capsules 1996	15-38	1.5-6.2	83-93	MAFF 1997b				
(n=4) ng TEQ/kg oil								
Halibut liver oil capsules 1996	5-9.5	1.7-6.1	63-68	MAFF 1997b				
(n=2) ng TEQ/kg oil								

Human exposure to dioxin-like PCBs

Table 2 lists the background tissue concentrations of dioxin-like PCBs for human milk, blood and adipose from several European and North American studies. The significance of dioxin-like PCBs in terms of human body burden is apparent, at between ~50 and 70% of Σ TEQ. Congener profiles in human tissues/blood appear similar in all the reviewed studies. PCB-TEQ in human milk fat is usually dominated by PCB-156 (23 - 36%), PCB-126 (22-37%) and PCB-118 (10-17%), which collectively constitute between 50% and 90% of the Σ PCB-TEQ. Hong et al. (1994) reported that PCB-118, PCB-126, PCB-105 and PCB-156 accounted for ~70% of Σ TEQ in US milk. This congener pattern is similar for both the UK, PCB-156, PCB-126 and PCB-118 (MAFF 1997c) and Finland, PCB-126, PCB-156 and PCB-118 (Vartiainen et al., 1997). Unfortunately, the limited data makes a comparison of congener patterns in older versus younger age groups (i.e. related to different types of exposure for example) very difficult.

Exposed populations as a result of elevated fish consumption have been identified in the literature. For example, a study by Cole et al. (1995) found that blood in fish eaters aged >50 years old contained double the PCB- and PCDD/F-TEQ compared to a non-fish eating group. Additionally, fish eaters had a 13% greater PCB-TEQ than non fish eaters. Dewailly et al., (1994) found elevated levels of dioxin-like PCBs in the breast milk of Inuit women in the Arctic, whose principal protein sources are fish and sea mammals. Nursing infants represent a sub-population which are also at increased risk in term of TEQ-exposure.

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A selective dietary accumulation and enrichment of the dioxin-like PCBs could represent an important effect occurring in the human foodchain, given the significance of dioxin-like PCBs measured in human tissues.

MEDIA	PCB-TEO	PCDD/F-	%PCB TEO	Reference
(i) Blood (ng TEO/g fat)	100 124	TFO	/01 05 1EQ	ACTO CALC
Fish-eaters/Non fish-eaters	17/97	34/37	34/24	Cole et al 1995
(Great Lakes)	111211	5 1157	5424	Colo et al., 1995
Yusho control	12	20	38	Masuda 1996
Iananese	28	28	50	Masuda 1996
Children (urban area)	9.6-12	7.3-8.2	57-59	Wuthe et al., 1994
Children (industrialised area)	9.1-11	9.0/10	51-53	Wuthe et al., 1994
Children (rural)	12.8-13.5	9.3-10	57-58	Wuthe et al., 1994
Background (Germany)	9.1	19	32	Päpke et al., 1996
Serum (Spain) *	7.03	15.7	31	Jimènez et al., 1996
(ii) Human adipose (pg TEQ/g fat)				
Adipose (UK) n= 5	116	57	67	Duarte-Davidson et al., 1993
Adipose (Germany)*	43	29	60	Körner et al., 1994
* PCB 126 and 169 only				
(iii) Human milk (pg TEQ/g fat)				
Milk (Germany)	31.7	16.7	65	Malisch 1996
Milk (Denmark)	17.2	16.7	51	Hilbert et al., 1996
Milk (WHO)	21	20.6	50	Ahlborg 1994
Milk (Japan)	96	58	58	Masuda 1996
Milk (UK)	10-12	21-24	32-35	MAFF 1997a
Milk (Sweden)	12-26	7-12	63-68	Dahl et al., 1995
Milk (Finland - urban) n=37	25	34	58	Vartiainen et al., 1997
Milk (Finland - rural) n=33	26	20	57	Vartiainen et al., 1997
Milk (Netherlands)	80	31	72	Tuinstra et al., 1994
Milk (USA) n=30	35	17	67	Hong et al., 1994

Table 2 PCB and PCDD/F concentrations (expressed as ΣTEQ in ng/kg lipid) for human tissues, together with the percentage contribution of ΣTEQ attributable to PCB

Concluding remarks

Dioxin-like PCBs represent an important component of the Σ TEQ in many environmental media. Specifically, in animal produce and in fish PCBs dominate the Σ TEQ ingested by humans. This in turn leads to high background body burdens in humans with PCB-TEQ greater than that associated with PCDD/Fs. High fish consumers are considered to be apparently subject to elevated TEQ exposure from dioxin-like PCBs. This has important implications for exposure assessment studies which have previously only been concerned with PCDDs and PCDFs. Unlike PCDD/Fs, dioxin-like PCBs are not controlled within the food chain. Sources and pathways of exposure are poorly defined. In this respect, it is important to determine the status of current sources releasing dioxin-like compounds into the environment, i.e. the relative contribution of controlled large industrial emission sources versus uncontrolled open sources such as small capacitors, lighting ballasts or building sealants. More research is needed to place these compounds in an integrated risk evaluation framework.

Acknowledgments

Ruth Alcock would like to thank the UK Environment Agency and NERC for supporting a Post-Doctoral Fellowship Award to investigate sources and fate of organic contaminants in the environment.

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