Depletion Rates of PCDDs in Bull Calf Tissues

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Introduction

In 1991 analysis of milk from individual farms near Bolsover, Derbyshire, UK, revealed a localized contamination hot-spot. One of the farms involved did not contribute milk to the public supply but had a suckler herd and sold on young animals for beef production. Milk from this farm was found to contain an extremely high level of 3.4 ng TEQ/kg of PCDDs and PCDFs (whole milk) and tissues from some calves raised on this farm also showed very high levels ranging from 2.5 to 6.9 ng TEQ/kg in muscle and 23.7 to 60.0 ng TEQ/kg in liver expressed on a whole sample basis.

To support the development of a management strategy for the incident a study was initiated to investigate the rate of change in concentrations of dioxins in the tissues of growing calves from this farm when fed on a diet containing only background levels of PCDDs and PCDFs.

Although a number of depletion studies have been reported they have dealt primarily with lactating animals. The only study of non-lactating animals was based on 3 adult females maintained at a relatively stable body-weight [1]. In this study sequential subcutaneous fat biopsy samples were obtained over a period of 220 days. The mean half-lives for different congeners were between 160 and 260 days.

Experimental

Five 9 month old bull calves (Table 1) were purchased, moved to an Experimental Husbandry Farm where they were placed on a diet that is assumed to have been essentially dioxin-free, and kept in indoor pens to minimise exposure to dioxins from non-dietary sources. Single calves were slaughtered at approximately monthly intervals after an initial two month period and samples of liver, muscle, subcutaneous fat, perirenal fat and either omental or peritoneal fat taken for analysis. Tissues were freeze-dried using the entire sample received by the laboratory. The dried material was thoroughly mixed and, in the case of muscle and liver, ground to a powder before sub-sampling.

After addition of $^{13}C_{12}$, internal standards, PCDDs and PCDFs were isolated by the method of Smith *et al* [2] using an automated apparatus (Fluid Management Systems) [3] and determined by HRGC/HRMS using a non-polar GC column. Further details are given elsewhere [4].

Results

Full results for a single animal are given, on a fat weight basis, in Table 2.

Prior to this study results on a small group of animals from the same farm had shown very high levels ranging from 2.5 to 6.9 ng TEQ/kg in muscle and 23.7 to 60.0 ng TEQ/kg in liver expressed on a whole sample basis. In the current study levels were somewhat lower, being in the range 0.5 to 1.0 ng TEQ/kg for muscle and 2.6 to 4.6 ng TEQ/kg for liver.

The objective in this experiment was to obtain information on the rate of depletion in the tissues of contaminated animals when switched to clean feed. The exact source of PCDDs and PCDFs in the Bolsover area has not been conclusively identified and the exact congener pattern involved as well as the route and timing of exposure of the animals are unknown. However, it is clear that 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 1,2,3,6,7,8-HxCDD were the dominant congeners and although other PCDDs and PCDFs were also elevated above background levels the difference is less marked. These congeners together with 2,3,4,7,8-PeCDF are also the most important toxicologically.

Results for different tissues showed uniformity between the fat stores but some considerable differences with respect to muscle and liver. Taking into account each of these congeners and all the animals, there was a general tendency for concentrations in muscle, expressed on a fat basis, to be lower than in the depot fat, in some cases by 50%. The concentrations of 2,3,7,8-TCDD in liver were comparable to the depot fat but this was not true of other congeners; for HxCDD the liver typically had a 4-fold higher concentration.

Although the different animals, representing different depletion times, showed some apparent differences in the distribution there did not appear to be a trend and we conclude that either experimental or biological variability, or both, are more important than time dependant changes. It is therefore reasonable to base examination of the depletion on the mean depot fat values.

Figures 1 to 3 show the concentrations of the selected congeners against time. Although there is a marked downward trend there is considerable scatter that cannot be explained by analytical variability. Because of the limited number of points and the apparent scatter it is not possible to estimate the coefficients of the expected exponential decay. The results for 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD are consistent with half-lives in the range of 100 to 200 days. The decline in 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDD appears to be considerably slower.

Each point represents just a single animal and the concentrations must have been influenced by both biological variability and possibly by differences in the starting concentrations of the different animals.

Since this study was performed on young animals, an increase in body weight occurred over the period of study and part of the decrease in PCDD concentrations must be attributable simply to growth. In a crude attempt to allow for this effect the mean fat concentrations were normalised to the initial weight of each animal. The results are shown in Figures 4 to 6. This simple treatment depends on the assumption that the weight increase is equally divided over different tissue types and this is unlikely to be true. It is however noteworthy that the concentrations of 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDD approximate to a steady state with this treatment.

References

1. M. Olling, P.L.M. Berende, H.J.G.M. Derks, A.K.D. Liem, H. Everts and A.P.J.M. de Jong, The toxicokinetics of PCDDs and PCDFs in non-lactating cows, RIVM Report 328904003, September 1991.
2. L.M. Smith, D.L. Stalling and J.L. Johnson, Anal. Chem., **56**, 1830 (1984).
3. W.E. Turner, S.G. Isaacs and D.G. Patterson Jr., Chemosphere, **25**, 805 (1992).
4. J.R. Startin, C. Wright M. Kelly and E.A. Charlesworth, Dioxin concentrations in the blood of individuals resident on farms near Bolsover, UK, Submitted for presentation at Dioxin 94, Kyoto, Japan.

Table 1. Details of animals studied.

Animal	Date of birth	Date of move	Date of slaughter	Depletion period (days)	Weight at start (kg)	Weight at slaughter (kg)
902	19/3/91	17/12/91	14/2/92	59	272	296
904	23/3/91	17/12/91	16/3/92	89	290	360
905	23/3/91	17/12/91	15/4/92	119	289	426
907	26/3/91	17/12/91	11/5/92	145	308	497
914	17/4/91	17/12/91	8/6/92	173	283	492

Table 2. PCDD/F Levels for Animal 902 (ng/kg fat).

	Liver	Muscle	Fat			
			Sub-cut.	Perirenal	Peritoneal	Mean
2,3,7,8-TCDD	20.0	17.9	34.3	31.2	27.8	31.1
1,2,3,7,8-PeCDD	36.9	17.0	32.0	28.9	23.9	28.3
1,2,3,4,7,8-HxCDD	*	*	*	*	*	*
1,2,3,6,7,8-HxCDD	352*	115*	173*	161*	144*	159*
1,2,3,7,8,9-HxCDD	151	25.6	42.2	38.4	34.6	38.4
1,2,3,4,6,7,8-HpCDD	60.7	11.4	6.3	5.7	5.7	5.9
OCDD	157	48.7	8.0	6.0	6.2	6.7
2,3,7,8-TCDF	0.6	<1.1	<0.5	<0.5	<0.5	<0.5
1,2,3,7,8-PeCDF	<0.5	<1.1	<0.5	<0.5	<0.5	<0.5
2,3,4,7,8-PeCDF	12.9	4.6	7.7	7.0	6.3	7.0
1,2,3,4,7,8-HxCDF	10.9	3.0	3.4	3.3	2.8	3.2
1,2,3,6,7,8-HxCDF	12.1	4.3	5.4	5.3	4.8	5.2
1,2,3,7,8,9-HxCDF	0.8	<1.2	<0.5	<0.5	<0.5	<0.5
2,3,4,6,7,8-HxCDF	17.7	4.9	5.8	5.8	5.3	5.7
1,2,3,4,6,7,8-HpCDF	11.5	3.6	2.7	2.5	2.3	2.5
1,2,3,4,7,8,9-HpCDF	1.4	<1.7	<0.5	<0.5	<0.5	<0.5
OCDF	3.1	<2.6	0.6	<0.5	<0.5	<0.5
Total TEQ	100	44.4	77.2	70.6	62.0	69.9

* indicates that 1,2,3,4,7,8-HxCDD was incompletely resolved from 1,2,3,6,7,8-HxCDD and it was not possible to make a separate measurement; the contribution to the combined concentration made by 1,2,3,4,7,8-HxCDD was small.

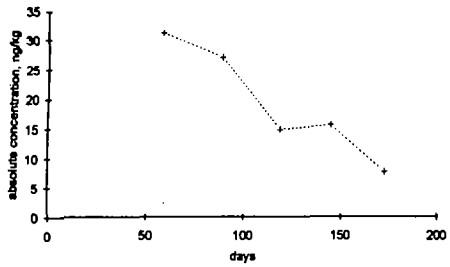

Figure 1. Relationship between depletion period and mean fat concentration of 2,3,7,8-TCDD.

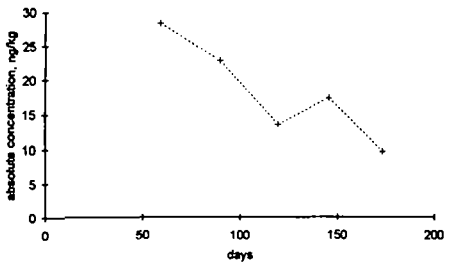

Figure 2. Relationship between depletion period and mean fat concentration of 1,2,3,7,8-PeCDD.

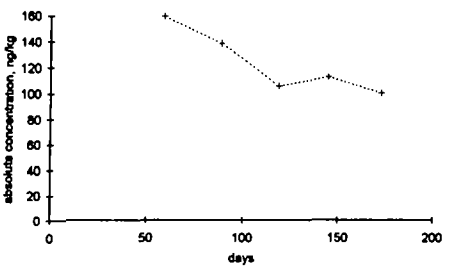

Figure 3. Relationship between depletion period and mean fat concentration of 1,2,3,4,7,8/1,2,3,6,7,8-HxCDD.

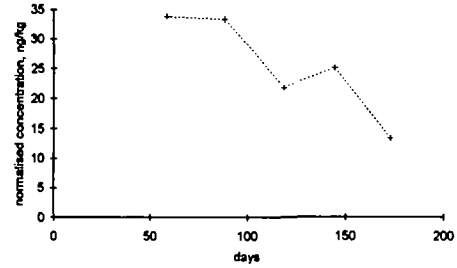

Figure 4. Relationship between depletion period and mean fat concentration of 2,3,7,8-TCDD normalised to body weight.

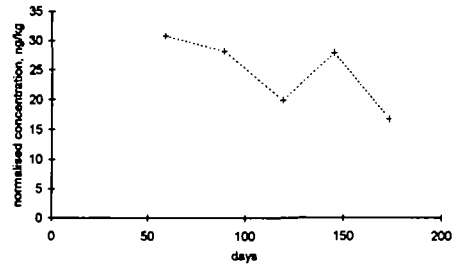

Figure 5. Relationship between depletion period and mean fat concentration of 1,2,3,7,8-PeCDD normalised to body weight.

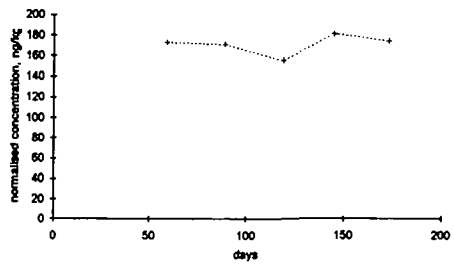

Figure 6. Relationship between depletion period and mean fat concentration of 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDD normalised to body weight.