THE FORMATION OF HEPTA- AND OCTA-DIOXINS IN FECES OF COWS FED PENTACHLOROPHENOL TREATED WOOD

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Introduction

Mass balances of polychlorinated dibenzo-*p*-dioxins (PCDDs) and dibenzofurans (PCDFs) in cows administered pentachlorophenol (PCP) treated wood indicated that excretion of HpCDD and OCCD in feces exceeded intake by factors of 1.7 and 3.4, respectively.[1] The initial hypothesis was that the apparent synthesis occurred in the rumen because this is a site of active microbial fermentation, and other fermentation systems had been implicated in the synthesis of HpCDD and OCDD.[2,3] This hypothesis was not confirmed when fermentation of PCP-treated wood with rumen microorganisms failed to yield HpCDD and OCDD in excess of the unfermented controls.[1] This report presents results of additional studies on the sites and circumstances of the apparent HpCDD and OCDD synthesis in cows fed PCP-treated wood.

Methods and Materials

A second mass balance study was conducted with two cows using the same PCP-treated wood. The animal management practices and dose rate were the same as in the previous study except that the dosing period was only 28 days instead of 56 days.[1,4] At the end of the mass balance measurements, the animals were slaughtered and samples of gastrointestinal tract contents were obtained. The samples were acidified to stop microbial activity. Another study conducted in which fresh cow feces were spiked with PCP-treated wood. Control samples were acidified immediately whereas the experimental samples were allowed to ferment at room temperature for 24 hours. Sample preparation and analyses for PCDD/Fs were carried out as previously reported.[4]

Results and Discussion

The mass balance measurements (Table 1) only partially confirmed the results of the previous study. Excretion of OCDD in feces was nearly double the intake, but excretion of HpCDD did not exceed intake. Since coefficients of variation of fecal excretion values are large[1], 20 to 30% deviations from 100% recovery in the case of a few congeners are not considered significant.

The PCDD/F concentrations in the contents of the rumen and ileum of animals slaughtered at the end of the dosing period are shown in Table 2. Concentrations in the diet were the value calculated by summing the PCDD/F intake from wood and feed, and dividing by total feed intake. Concentrations in gastrointestinal tract contents and feces were adjusted for disappearance of dry matter using several heavy metals as markers. Adjusted concentrations of PCDD/Fs in the rumen did not differ significantly from the concentrations in the diet, confirming the lack of synthesis of OCDD at this site in previous *in vitro* studies.[1] The adjusted concentrations of PCDD/Fs in the ileum were somewhat lower than in the diet, but the ratio of OCDD to other congeners had not

Table 1. Fractional disposition of the daily dose of PCDDs and PCDFs in two cows administered pentachorophenol-treated wood for 28 days[a]

		Recovery, %			
Congener	Intake, pg/d	Storage[b]	Milk	Feces	Total
1,2,3,4,7,8-HxCDD	11,700	15.2	31.7	81.3	128.2
1,2,3,6,7,8-HxCDD	114,000	16.5	34.5	80.1	131.1
1,2,3,7,8,9-HxCDD	24,600	10.2	25.9	107.2	143.2
1,2,3,4,6,8,9-HpCDD	3,300,000	3.3	4.9	91.1	99.2
1,2,3,4,6,7,8,9-OCDD	16,500,000	2.4	0.9	183.8	187.1
1,2,3,4,7,8-HxCDF	9,900	12.8	32.1	83.1	128.0
1,2,3,6,7,8-HxCDF	12,000	6.8	31.4	123.4	161.5
2,3,4,6,7,8-HxCDF	114,000	10.2	21.4	70.8	102.3
1,2,3,4,6,7,8-HpCDF	660,000	1.9	4.6	99.1	105.6
1,2,3,4,7,8,9-HpCDF	30,000	4.3	7.3	74.7	86.4
1,2,3,4,6,7,8,9-OCDF	3,900,000	0.5	0.4	96.7	97.6

[a] Congeners not listed are those with concentrations below the limit of quantitation in one or more matrices and the PCDFs that are metabolized.[4]

[b] Storage in body fat was estimated by measuring concentrations in perirenal fat and multiplying by the estimated fat content of the animal.[5] Storage also includes estimates for liver, which was analyzed separately.

changed indicating that synthesis had not occurred to this point in the gastrointestinal tract. In agreement with results in Table 1, OCDD concentrations in feces were significantly enhanced.

Fecal incubation studies were carried out to determine if HpCDD and OCDD synthesis occurred post excretion. The results (Table 3), although not dramatic, demonstrated increased concentrations of OCDD after incubation for 24 h at ambient temperatures. This finding, together with the negative results of the gastrointestinal tract analyses, suggests the probability that the observations in the mass balance studies involved synthesis of the compounds after excretion while the feces were in the barn prior to collection and processing. This time period could be as great as 24 h and it would also include several addition hours while samples were processed and while drying temperatures rose to the point that would destroy enzymes and microorganisms.

The significance of the observations from these studies and related studies of sewage treatment[2] and composting[3] is difficult to evaluate because of the highly variable results. The finding provide an explanation for the common conclusion that OCDD is much more prevalent in the environment than can be accounted for by introductions from known sources. However, the toxicity equivalence of HpCDD and OCDD are low so that the potential additions of these congeners to the environment may not be of great human health significance.

Table 2. Relative concentrations of PCDDs and PCDFs in feed, intestinal tract contents, and feces of two cows administered pentacholorphenol-treated wood[a]

	Diet	Rumen	Ileum	Feces
Congener		pg/g		
1,2,3,7,8-PeCDD	0.25	0.24	0.26	0.14
1,2,3,4,7,8-HxCDD	0.62	0.76	0.78	.50
1,2,3,6,7,8-HxCDD	5.8	5.0	3.6	4.6
1,2,3,7,8,9-HxCDD	1.3	1.5	1.1	1.4
1,2,3,4,6,7,8-HpCDD	167	136	92	152
1,2,3,4,6,7,8,9-OCDD	860	940	620	1540
1,2,3,7,8-PeCDF	0.10	0.08	0.13	0.04
2,3,4,7,8-PeCDF	0.10	0.10	0.14	0.06
1,2,3,4,7,8-HxCDF	0.66	0.60	0.61	0.48
1,2,3,6,7,8-HxCDF	0.67	0.62	0.71	0.80
2,3,4,6,7,8-HxCDF	0.68	0.69	0.82	0.48
1,2,3,4,6,7,8-HpCDF	34	37	23	34
1,2,3,4,7,8,9-HpCDF	1.6	1.4	1.1	1.2
1,2,3,4,6,7,8,9-OCDF	197	202	131	190

[a] Values are averages of two cows. Congeners not listed had concentration below the detection limits in one or more samples. Concentrations in diet were calculated from the measured feed intake and the concentration in wood.[4] Concentrations in gastrointestinal tract contents and feces were corrected for dry matter disappearance, and the mass added by acidification-neutralization, to the original weight of dry matter represented in the diet using Zn, Cu, Mn, and Sr as markers.

Several areas require further study to better understand and evaluate the significance of the apparent synthesis of OCDD and possibly other PCDDs. Since the colon was not sampled in our work, this site can not be ruled out a possible site of synthesis. The liver as a site of synthesis should be evaluated because there is evidence that synthesis can occur if the appropriate precursors are present.[6] The precursors of HpCDD and OCDD are not known, but dimers like nonachloro-2-phenoxyphenol can be converted to OCDD in abiotic and biological systems.[6,7] If less chlorinated dimers are present as contaminants, conversion to the analogous PCDDs with higher toxic equivalence could be significant.

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Table 3. Concentrations of PCDDs and PCDFs in feces spiked with pentachlorophenol-treated wood and incubated at ambient temperatures for 24 h[a]

Congener	Control, pg/g	Incubated, pg/g
1,2,3,7,8-PeCDD	1.1	1.2
1,2,3,4,7,8-HxCDD	2.6	2.6
1,2,3,6,7,8-HxCDD	23.7	24.5
1,2,3,7,8,9-HxCDD	7.1	6.9
1,2,3,4,6,7,8-HpCDD	610	750
1,2,3,4,6,7,8,9-OCDD	7,100	8,340
2,3,7,8-TCDF	0.43	0.44
1,2,3,7,8-PeCDF	1.1	1.0
2,3,4,7,8-PeCDF	1.5	1.7
1,2,3,4,7,8-HxCDF	5.2	5.6
1,2,3,6,7,8-HxCDF	5.0	5.2
2,3,4,6,7,8-HxCDF	4.1	4.4
1,2,3,7,8,9-HxCDF	0.65	0.69
1,2,3,4,6,7,8-HpCDF	160	160
1,2,3,4,7,8,9-HpCDF	7.1	7.8
1,2,3,4,6,7,8,9-OCDF	900	890

[a] Congeners not listed had concentrations below the detection limit. The control was spiked feces acidified to prevent microbial action. Concentrations in the control were adjusted for the weight added by acidification and neutralization based on the dilution of Zn, Cu, Ni, Mn, Fe, and Sr.