Environ. Sci. Technol. **2006**, *40,* 1462−1468

Congener-Specific Tissue Distribution and Hepatic Sequestration of PCDD/Fs in Wild Herring Gulls from Bohai Bay, North China: Comparison to Coplanar PCBs

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Tissue distribution is an important property of pharmacokinetic behaviors of dioxins to provide information for risk assessment to wild avian species. In this study, concentrations of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and coplanar polychlorinated biphenyls (coplanar PCBs) were determined in muscle, liver, spleen, kidney, brain, and adipose of wild herring gulls collected from Bohai Bay, North China. Tissue distribution results showed preferential accumulation of PCDD/Fs in liver and of co-PCBs in adipose. The congener patterns of coplanar PCBs were constant in different tissues, but the congener patterns for PCDD/F were tissue-specific. The liver/adipose concentration ratios for PCDD/Fs were found to increase statistically significantly with log K_{ow}, providing the quantitative relationship of structure−activity for hepatic sequestration of PCDD/Fs for the first time. Furthermore, this relationship was compared with those developed on the basis of previous results reported in the literature showing that the wild herring gulls in Bohai Bay are still in the exposure period.

Introduction

Planar halogenated aromatic hydrocarbons (PHAHs) such as polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and non- and mono-ortho polychlorinated biphenyls (non- and mono-ortho PCBs) are ubiquitous, persistent, and highly lipophilic environmental contaminants. There is growing evidence that these compounds can accumulate in animal body tissues through the aquatic food web (*1*−*3*) and cause a wide range of toxic and biological effects such as reproductive failure, immune deficiency, teratogenesis, and abnormal behavior in animals and humans (*4*−*6*).

To assess the ecotoxicological risks to the high trophic level animals, toxic equivalency quantifications (TEQs) are always calculated using the World Health Organization (WHO) toxic equivalency factors (TEFs) (*7*). The present TEF concept is based on the dose-additivity model for Ah receptor-mediated responses, and the importance of pharmacokinetic behaviors has been proposed (*7*−*10*). Tissue distribution is an important property of pharmacokinetic behaviors of pollutants (*11*) and is generally investigated to provide information for the development of pharmacokinetic models (*12*, *13*). The tissue distribution of dioxin in biota has been extensively studied in laboratory experiments using rodents (*14*). However, surprisingly few studies have investigated tissue distributions of dioxins in birds, although birds have separate sets of TEF values. An early publication by Braune et al. (*15*) reported the increasing degree of selective storage in liver with the degree of chlorination of PCDD/Fs in captive herring gulls. A similar observation (*16*) was also made in a later laboratory experiment, which found that liver/adipose concentration ratios in chickens increase from the lower to the higher chlorinated PCDD/Fs during the exposure period, and the possible reason was hepatic sequestration of dioxin-like isomers by binding to hepatic microsomal proteins such as cytochrome P450 (*6*, *17*). Of the limited three papers describing tissue distribution and toxicokinetic behaviors of dioxins in wild birds (*17*−*19*), Kubota et al. first used liver/muscle concentration ratios to describe hepatic sequestration (*17*). On the basis of Kubota's data, the liver/muscle concentration ratios of PCDD/Fs did not increase with the degree of chlorination, a result different from that in the laboratory (*17*). As for coplanar PCBs, even less information is available with regards to their tissue distribution (*19*).

In Bohai Bay, herring gulls are the top predators of the marine food web and addressed as a suitable bioindicator species in local ecosystems (*3*, *20*). To provide information of pharmacokinetic behaviors for PCDD/Fs and coplanar PCBs in wild herring gulls to better evaluate the TEFs in birds, this study presents the results from analysis of 9 PCDDs, 11 PCDF isomers, and 12 non-, mono-ortho PCB congeners and their distributions in samples at 6 anatomical sites: muscle, liver, spleen, kidney, brain, and adipose of six herring gulls collected from Bohai Bay. The relationships between the liver/adipose concentration ratios and log K_{ow} for dioxin isomers were analyzed for the first time to compare the hepatic sequestrations to PCDDs, PCDFs, and coplanar PCBs.

Materials and Methods

Sample Collection. Herring gulls (*Larus argentatus*) used in this study were long inhabitants of Tianjin, North China, with a relatively large population, and six birds were captured in November 2002 prior to their winter migration on the coast of Bohai Bay (39°07′ N, 117°44′ E). The body length and weight of the birds were 35 cm (31−41) and 211 g (142−321 g), respectively. According to the pattern and color of bird body plumage and beak color (Supporting Information Figure S1) (*21*−*23*), the collected herring gulls were identified to be the third-winter ones. Because the hatching time of the birds was from April to June (*24*), the age of the birds collected in November were determined to be about 30 months. Although we cannot identify sex of the birds, the differences of accumulation and pharmacokinetic behaviors between male and female birds can be neglected, on the basis of the fact that the fertilization for Herring gull species often starts at fourth-winter (*21*−*23*) and male−female difference in ex-

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10.1021/es052249s CCC: $33.50 © 2006 American Chemical Society
Published on Web 01/25/2006

posure level was due to maternal transfer after maturity of the animals (*6*).

Chemical Analysis. Dioxin analysis was conducted by following the method described in detail elsewhere (*3*). The tissue samples were freeze-dried and spiked with a mixture of ^{13}C-labeled PCDDs, PCDFs, and non- and mono-ortho PCBs internal standards. Then the spiked samples was refluxed for about 15 h in 1 N KOH−ethanol and the solution was transferred to a separatory funnel containing 500 mL of pure water, 100 mL of hexane, and about 20 g of NaCl. After the solution was shaken and partitioned, the hexane layer was collected. This procedure was repeated three times, and the hexane solution was concentrated and treated with sulfuric acid (2 times) in a separatory funnel. Then the hexane layer was rinsed with pure water and dried by passing through anhydrous sodium sulfate (Kanto Chemical Co., Inc.) in a glass funnel. The solution was concentrated to about 2 mL and sequentially subjected to multilayer silica gel and activated carbon-impregnated silica gel column chromatography. Extracts were passed through a multiplayer silica gel packed glass column (2 g of silica gel impregnated with $AgNO_3$ (10% mass), 0.5 g of silica gel (silica gel 60, Metocean Co.), 5 g of silica gel impregnated with sulfuric acid (50% mass), 0.5 g of silica gel, 2 g of silical gel impregnated with KOH (2% mass), and 0.5 g of silica gel in sequence) and eluted with 200 mL of hexane. The hexane extract was concentrated, passed through activated carbon-impregnated silica gel column (1 g of activated carbon dispersed silica gel (Kanto Chemical Co., Inc.)), and eluted with 25 mL of hexane as a first fraction, which did not contain dioxin. The second fraction eluted with 40 mL of 25% DCM/hexane (v/v) containing mono-ortho PCBs. The third fraction was eluted with 50 mL of toluene from the other end of the column containing PCDD/Fs and non-ortho PCBs. An aliquot of the third fraction was spiked with 1,3,6,8-TeCDF for analysis of PCDD/Fs, and aliquots of the last two fractions were mixed and spiked with 2,2′,5,5′-TeCB for analysis of mono- and non-ortho PCBs. The instrumental conditions of dioxin analysis, the quantitation and quality assurance quality control (QA/QC), and the lipid content analysis are provided in the Supporting Information.

Statistical Analysis. The normality of the data was tested using the Shapiro−Wilk tests (sample size: $3 < n < 50$). A log-transformation was done to ensure the normality of the data distribution in all tissues. A "one way analysis of variance (ANOVA)" was performed to look for differences in data between the tissues. The Levene test was used to check the equality of variances because log transformations did not normalize all the data including TEQ values and proportions of different isomers. Where variances were equal, the difference of data was analyzed by the *F* test. Where the equality of variances could not be assumed, Welch and Brown-Forsythe's robust test for the equality of means was used. Once the difference between means was assumed, multiple paired comparisons were used to determine which means differed from one another. The Tukey HSD was used where variances were presumed equal, and the Games−Howell test was used where equality of variances could not be assumed.

The correlations between concentration ratios of liver/adipose, log K_{ow} values were examined by Spearman's rank correlation test, and when the value of *p* was below 0.05, the linear regression was regarded as significant. The software used was SPSS 11.0 (SPSS Inc., Chicago, IL).

Results and Discussion

Concentrations and Isomer Patterns. Table 1 lists the PCDD/F concentrations in the different tissues of herring gulls collected from Bohai Bay. The sum concentration of 20 PCDD/Fs in brain (3.3 ± 1.8 pg/g ww (wet weight)) was

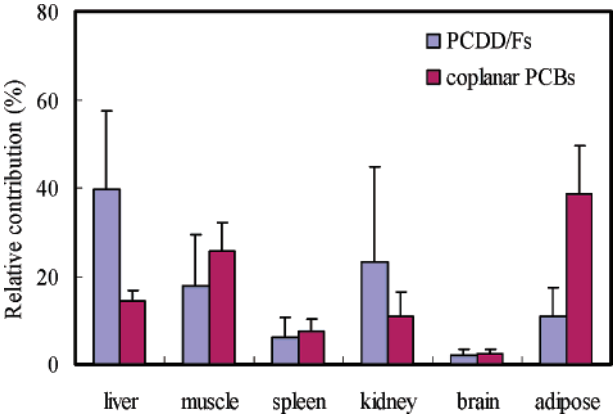

FIGURE 1. Relative contribution (percent) of PCDD/Fs and coplanar PCBs in different tissues to the total body concentrations (lipid corrected) of herring gulls collected in Bohai Bay.

significantly lower than those in other tissues ($p < 0.05$), and that in adipose (156 ± 74.5 pg/g ww) was significantly higher than those in other tissues ($p < 0.05$) except for liver (78.1 ± 60.0 pg/g ww). While no significant concentration differences were found among muscle (26.7 ± 8.1 pg/g ww), spleen (13.6 ± 6.6 pg/g ww), and kidney (55.0 ± 95.9 pg/g ww), the concentration in liver was significantly higher than that in spleen ($p < 0.05$). When the values were expressed on a lipid weight basis, the sum concentration in brain was still significantly lower than those in other tissues ($p < 0.05$) except for spleen. The concentration in adipose became significantly lower than that in liver ($p < 0.05$), and that in spleen was found significantly lower than those in liver, muscle, and kidney ($p < 0.05$) (Figure 1). Concentrations of coplanar PCBs were apparently higher than those of PCDD/Fs in all tissues, and tissue distributions of coplanar PCBs differ greatly from those of PCDD/Fs. When the concentrations were measured on a wet weight basis, the sum concentration of coplanar PCBs in adipose (100 000 ± 121 000 pg/g ww) was significantly higher than those in other tissues ($p < 0.05$), and that in brain (507 ± 299 pg/g ww) was significantly lower than those in liver (3600 ± 3500 pg/g ww), muscle (5300 ± 3900 pg/g ww), and spleen (2100 ± 1300 pg/g ww) ($p < 0.05$). Significantly different concentration was also found between muscle and kidney (1400 ± 637 pg/g ww) ($p < 0.05$). When the amounts of contaminants were based on lipid weight, the concentration in adipose was only significantly higher than those in kidney, spleen, and brain, and the concentration in brain was significantly lower than those in liver, muscle, and kidney ($p < 0.05$). The concentration in muscle was significantly different from that in spleen ($p < 0.05$) (Figure 1). The major distribution differences between PCDD/Fs and coplanar PCBs were that PCDD/Fs were mainly deposited in liver, and coplanar PCBs were mostly in adipose, which will result in different trophic magnification factors (TMFs) for these chemicals when concentrations from different organs are used to correlate with trophic levels. The minimum concentrations of PCDD/Fs and coplanar PCBs were all found in brain tissue, which is similar to results of other studies (*18*). This could be because the dioxin is hindered from crossing the blood/brain barrier and/or because of the polar lipid composition of the brain (*25*), since brain tissue consists mainly of relatively polar lipids (e.g., polarphospholipids) (*26*, *27*) compared with triacylglycerol-rich tissues, and these polarphospholipids constitute unfavorable sites for organochlorine accumulation (*28*). In kidney tissues, the concentrations of dioxins were found to be relatively high in two eagles collected from the upper Peninsula of Michigan (*19*). In this study while higher concentrations of PCDD/Fs in kidney than in muscle were determined in two birds, it was vice versa for another four birds. Concentrations of coplanar

TABLE 1. Concentrations (pg/g Wet wt) of PCDD/Fs and Non- and Mono-Ortho PCBs in the Different Tissues, Liver/Adipose Ratios, and Liver/Muscle Ratios of Herring Gulls Collected in Bohai Bay[a]

	tissue (lipid %)													
	liver (9.1 ± 2.6)		muscle (10.0 ± 4.7)		spleen (15.7 ± 2.7)		kidney (7.5 ± 3.9)		brain (10.9 ± 2.2)		adipose (89.1 ± 6.2)			
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	liver/adipose ratios[b]	liver/muscle ratios[b]
1,3,6, 8-TeCDD	0.43	0.21	0.22	0.23	1.15	1.11	2.4	2.1	0.45	0.46	1.8	2.4	3.7 (0.23−0.38)	2.1 (0.11−4.4)
1,3,7,9-TeCDD	0.13	0.12	0.07	0.1	0.24	0.39	1.1	1.1	0.14	0.12	0.64	0.81	3.6 (0.24−1.3)	4.0 (0.74−14.9)
2,3,7,8-TeCDD	1.2	1.0	1.03	0.45	0.46	0.49	1.0	0.89	0.41	0.28	8.8	4.5	1.4 (0.60−4.3)	1.0 (0.76−1.6)
TeCDDs	1.6	1.0	1.3	0.69	1.85	1.69	4.2	3.1	1.0	0.79	11.5	6.3		
1,2,3, 7,8-PeCDD	2.7	3.6	1.4	0.72	0.83	1.0	1.0	0.8	0.05	0.09	12.3	6.1	2.3 (0.76−9.4)	1.5 (0.87−3.1)
PeCDDs	2.7	3.6	1.4	0.72	0.83	1.0	0.87	0.90	0.04	0.09	12.3	6.1		
1,2,3, 4,7,8-HxCDD	1.9	1.6	0.40	0.12	0.19	0.33	0.14	0.16	ND	na	3.7	1.25	5.4 (0.95−16.2)	4.6 (0.61−9.2)
1,2,3, 6,7,8-HxCDD	4.9	5.7	1.13	1.14	0.33	0.46	0.34	0.34	ND	na	11.8	13.3	6.3 (0.89−27.7)	4.1 (1.7−11.3)
1,2,3, 7,8,9-HxCDD	0.72	0.63	0.24	0.18	ND	na	0.08	0.13	ND	na	2.8	2.3	3.2 (1.3−10.7)	3.6 (1.7−10.8)
HxCDDs	7.8	7.8	2.0	1.30	1.2	1.2	0.64	0.53	0.14	0.22	19.9	18.0		
1,2,3, 4,6,7, 8-HpCDD	8.0	5.8	2.4	1.32	0.32	0.38	2.4	4.3	0.20	0.30	5.3	5.7	32.0 (2.2−112.5)	4.2 (0.45−7.8)
HpCDDs	8.0	5.7	2.5	1.34	0.32	0.38	3.7	6.9	0.25	0.42	5.5	6.1		
OCDD	14.8	13.6	6.1	4.04	3.4	3.0	36.9	74.4	0.97	0.82	3.9	2.5	49.6 (2.7−129.8)	3.3 (0.26−6.1)
tot. PCDDs	34.7	27.5	13.5	4.6	7.6	5.1	38.7	74.3	2.4	1.6	53.3	33.5		
1,2,7, 8-TeCDF	0.06	0.07	0.06	0.05	0.27	0.20	0.35	0.30	0.13	0.12	0.43	0.56	1.1 (0.06−3.3)	1.0 (0.18−2.4)
2,3,7, 8-TeCDF	0.56	0.36	0.70	0.45	0.34	0.19	0.42	0.38	0.07	0.07	5.3	2.8	1.0 (0.46−2.2)	1.1 (0.17−2.2)
TeCDFs	3.0	1.4	5.3	2.7	2.1	1.6	3.0	3.1	0.71	0.54	32.8	14.4		
1,2,3, 7,8-P eCDF	0.7	0.50	0.41	0.27	0.19	0.19	0.05	0.09	ND	na	3.7	1.7	1.9 (0.03−4.7)	7.1 (0.02−34.1)
2,3,4, 7,8-P eCDF	6.7	6.7	1.8	0.66	1.8	1.4	1.2	0.77	ND	na	16.4	6.5	4.8 (1.3−18.7)	4.5 (0.45−15.9)
PeCDFs	10.4	8.0	4.4	1.9	2.5	1.7	1.9	1.06	0.10	0.10	41.1	18.8		
1,2,3, 4,7,8-HxCDF	9.9	12.3	0.69	0.36	0.56	0.65	0.69	0.46	ND	na	8.6	5.0	13.3 (2.2−56.2)	13.1 (3.1−42.5)
1,2,3, 6,7,8-HxCDF	8.7	10.1	0.56	0.32	0.29	0.50	0.26	0.25	ND	na	8.9	5.6	13.4 (2.4−58.4)	14.0 (4.0−43.3)
1,2,3, 7,8,9-HxCDF	ND	na	ND	na	ND	na	ND	na	ND	na	ND	na	9.4 (7.3−11.3)	1.0 (0.7−1.5)
2,3,4, 6,7,8-HxCDF	1.6	1.3	0.20	0.07	ND	na	0.26	0.26	ND	na	2.2	0.9	11.5 (1.8−48.0)	10.2 (2.0−33.6)
HxC DFs	22.0	23.2	2.8	0.92	0.89	1.2	1.2	0.94	ND	na	26.6	12.6		
1,2,3, 4,6,7, 8-HpCDF	4.6	3.4	0.42	0.14	0.15	0.16	1.0	2.1	ND	na	1.8	0.84	29.4 (8.2−91.8)	13.4 (2.0−26.6)
1,2,3, 4,7,8, 9-HpCDF	0.33	0.12	ND	na	ND	na	ND	na	ND	na	0.43	0.41	21.3 (3.8−83.6)	11.5 (2.0−28.0)
HpCDFs	5.0	3.4	0.51	0.23	0.12	0.15	3.7	8.5	ND	na	3.0	1.1		
OCDF	2.4	1.6	0.40	0.28	0.40	0.69	7.7	16.4	ND	na	0.57	0.49	139.8 (21.8−421.0)	24.1 (1.0−112.5)
tot. PCDFs	43.3	34.9	13.4	5.5	5.9	3.9	16.1	20.8	0.91	0.54	103	40.5		
tot. (PCDDs + PCDFs)	78.1	60.0	26.7	8.1	13.6	6.6	55.0	96	3.3	1.8	156	74.5		
3,3,4,4-TeCB	58.5	27.1	255	323	68.4	55.9	73.9	122	13.5	3.1	1200	528	0.47 (0.30−0.81)	0.52 (0.04−1.01)
3,4,4 ,5-TeCB	12.4	6.5	25.1	18.4	9.9	4.3	7.1	5.7	3.1	1.7	196	90.0	0.67 (0.37−1.6)	0.61 (0.17−1.13)
3,3,4,4,5-PeCB	31.7	74	50.3	22.9	22.8	16.0	17.6	15.0	4.3	2.6	598	330	0.53 (0.20−0.75)	0.63 (0.31−0.96)
3,3,4,4,5, 5-HxCB	19.0	87	14.2	6.8	6.5	5.9	4.7	2.8	0.21	0.34	136	57.7	1.2 (0.54−2.2)	1.32 (0.53−2.74)
tot. non-ortho PCBs	188	159	347	374	108	60.9	126	147	20.4	4.9	2200	508		
2,3,3 ,4,4-PeCB	755	871	1100	944	430	268	273	105	108	64.2	21000	27000	0.38 (0.29−0.45)	0.59 (0.30−0.92)
2,3,4,4,5-PeCB	63.0	64.6	99.0	82.2	36.7	21.7	21.5	13.8	10.2	5.9	1800	2100	0.38 (0.31−0.44)	0.58 (0.28−0.80)
2,3,4 ,4,5-PeCB	2000	2300	2800	2000	1100	759	765	307	285	171	61000	8000	0.38 (0.30−0.49)	0.62 (0.30−1.07)
2,3,4,4,5-PeCB	60.8	46.2	101	57.5	41.2	27.8	28.0	16.6	11.1	6.3	1700	1700	0.38 (0.30−0.47)	0.57 (0.31−0.78)
2,3,3,4,4,5-HxCB	223	178	363	222	133	93.5	96.3	36.2	32.3	20.8	7300	7200	0.35 (0.27−0.47)	0.57 (0.30−0.85)
2,3,3,4,4,5-HxCB	68.0	47.3	108	62.3	42.8	33.3	27.6	18.4	10.9	7.3	2000	1700	0.36 (0.29−0.46)	0.60 (0. 30−0.96)
2,3,4,4,5,5-HxCB	153	110	238	107	101	82.4	67	62	29.6	26.5	3800	3000	0.41 (0.26−0.56)	0.57 (0.31−0.81)
2,3,3,4,4,5,5-HpCB	52.4	51.5	91.7	66.8	31.6	30.4	26.8	30.3	8.6	8.2	1200	1000	0.42 (0.26−0.61)	0.50 (0.31−0.86)
tot. mono-ortho PCBs	3400	3500	5000	3600	1900	1300	1300	580	494	306	99000	120000		
tot. Co-PCBs	3600	3500	5300	3900	2100	1300	1400	637	507	299	100000	121000		
tot. TEQ	20.8	13.5	25.7	19.6	10.3	5.1	9.65	9.67	1.9	0.67	190.8	37.4		

[a] ND: nondetected. na: no value. [b] Concentrations below the detection limit were treated as half of the detection limit; the detection limits of TeCDD/F and PeCDD/F congeners, HxCDD/F, HpCDD/F, non- and mono-ortho PCB congeners, and OCDD and OCDF congeners were 0.02, 0.04, and 0.1 pg/g wt, respectively.

PCBs in kidneys of the six birds were all comparable with those in liver tissues. To our knowledge, this is the first report of retention of dioxin in the spleen of birds, and dioxins did not deposit preferentially in this organ.

Relative distribution patterns for PCDD/F congeners in liver, muscle, spleen, kidney, brain, and adipose are shown in Figure 2. OCDD was the predominant congener in spleen (12−47%) and brain (16−45%). In liver and muscle, proportions of OCDD were 6−29% and 5−36%, respectively, due to the low value in two birds, and in kidney, proportions of OCDD ranged from 14 to 68% in five birds but only 1% in one birds. This isomer has also been found to be abundant in sediment samples (OCDD: 56−91%) collected in Bohai Bay (*29*). Besides the above differences of isomer patterns among tissues, tissue-specific distribution of PCDD/F congeners was also found based on the ANOVA analysis. For example, the proportions of 1,2,3,7,8,9-HxCDD and 2,3,4,6,7,8-HxCDF in spleen (0.1−0.4%) were significantly different from those in liver (0.6−2.7%), muscle (0.4−2.4%), brain (0.4−1.8%), and adipose (0.8−2.5%) ($p < 0.05$) and the proportion of 2,3,4,7,8-PeCDF in brain (0.2−0.9%) was significantly lower than those in liver (4.6−11%), muscle (3.3−13%), spleen (9−20%), and adipose (4.4−22%) ($p < 0.05$) (Figure 2). It should be noted that the proportion of OCDD in adipose (1.5−5.3%) was significantly lower than those in other tissues (4.5−47%) ($p < 0.05$); the proportion of OCDF (0.03−1%) in adipose was significantly lower than those in liver (1.5−6.2%), kidney (0.2−15%), and brain (1−20%) ($p < 0.05$). The proportion of 1,2,3,4,6,7,8-HpCDD in adipose (1.3−5.5%) was significantly lower than that in liver (3.1−17%) ($p < 0.05$), and the proportions of 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD in adipose (3.6−13%) were significantly higher than those in liver (0.6−5.3%) and brain (0.2−15%) ($p < 0.05$) (Figure 2). The explanation may be that highly chlorinated PCDD/Fs do not

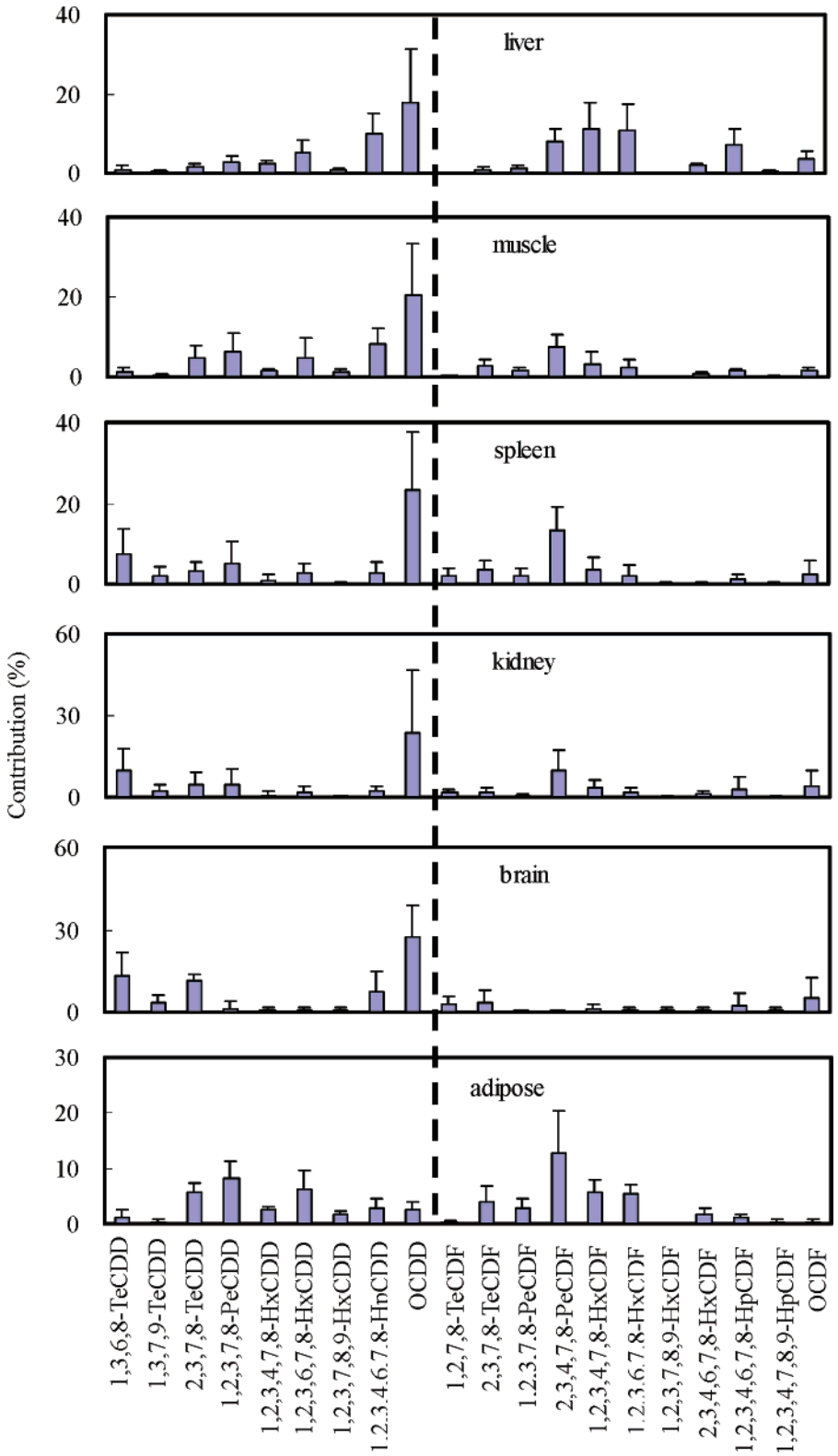

FIGURE 2. Relative contribution (percent) of PCDD/F congeners to total PCDD/F concentrations in different tissues of herring gulls.

accumulate preferentially in adipose due to the hepatic microsomal protein (*6*, *17*, *30*, *31*), and thus, lowly chlorinated isomers tend to deposit and showed a relatively high proportion.

Congener-specific analysis for coplanar PCBs showed constant distribution in different tissues (Supporting Information Figure S2). Among non-ortho PCBs, PCB-77 was the most predominant (44−69%) isomer in all tissues followed by CB-126 (16−28%), CB-81 (8−15%), and CB-169 (1−12%), which differed from the patterns reported for common cormorants in Lake Biwa, Japan, and Baikal seal (*Phoca sibirica*) from Lake Baikal, Russia (*6*, *17*). Among mono-ortho PCBs, congener patterns in different tissues were very similar to PCB 118 as the predominant isomer (57−59%) followed by CB-105 (21−22%), CB-156 (7−8%), and CB-167 (4−5%), and the congener patterns of mono-ortho PCBs have also been reported in different biological species from different areas (*2*, *6*, *17*, *32*, *33*). The lack of selectivity in intertissue

ratios for coplanar PCBs has also been reported for common PCBs and other organochlorines in captive herring gulls, indicating that, unlike PCDD/Fs, tissue distributions of these compounds were controlled principally by transport and equilibration among lipid pools (*15*).

TEQs. The relative toxic potential of PCDDs, PCDFs, and coplanar PCBs in tissues was calculated using the WHO toxic equivalency factors (TEFs) for birds (*7*). The concentration of TEQ in brain tissues (17±4 pg/g of lipid weight) was significantly lower than those in other tissues except for kidney (140±130 pg/g lw) ($p < 0.05$), and that in spleen (68±28 pg/g lw) was significantly lower than those in muscle (270 ± 200 pg/g lw) and adipose (220 ± 41 pg/g lw) ($p < 0.05$). No significant difference exists among TEQ concentrations in liver (230 ± 170 pg/g lw), muscle, kidney, and adipose ($p > 0.05$). The contributions of PCDDs to total TEQs in liver, muscle, adipose, spleen, kidney, and brain were 11−29%, 2−31%, 7−17%, 4−28%, 1−32%, and 11−32%, respectively, and those of PCDFs were 18−58%, 5−20%, 7−20%, 14−29%, 10−51%, and 2−14%, respectively. It should be noted that while non-ortho PCBs in muscle (59−92%), adipose (64−78%), spleen (53−80%), and brain (65−77%) are predominant contributors of total TEQ, its contributions in liver and kidney were in the wide range from 13 to 64% and from 3 to 76%, respectively.

Congener-Specific Hepatic Sequestration. To describe the distribution kinetics of PCDD/Fs in rats, liver/adipose ratios have been used as an important factor in some physiologically based pharmacokinetic (PB-PK) models (*34*, *35*). In this study, the average liver/adipose concentration ratios of PCDD/Fs and co-PCBs on a lipid weight basis for individual congeners were used, as shown in Table 1 and Figure 3. The results revealed that the liver/adipose ratios for PCDD/Fs were all higher than 1, and those for coplanar PCBs were all lower than 1, indicating that all the PCDD/Fs were primarily deposited in liver and the tissue selection for the trophic-transfer analysis of high chlorinated isomers would largely influence their TMF values. In addition, the liver/adipose ratios for PCDD/Fs were found to increase with their log K_{ow} values, of which the trend was similar to those of laboratory experiments for PCDD/Fs during the exposure period but different from those during the depuration period (*16*) (Figure 3a,b). This may indicate that herring gulls in Bohai Bay are still in the exposure period, which can be also supported by the relative consistency of PCDD/Fs concentrations throughout the entire sediment core in Bohai Bay by recent investigations (*25*). Finally, significant linear relationships can be established between liver/adipose ratios for PCDD/Fs and log K_{ow} values either in the present study (log liver/adipose ratios = 0.6461 × log K_{ow} − 3.9187, r^2 = 0.6778, $p < 0.001$) or in laboratory experiment (an exposure period of 164 days, log liver/adipose ratios = 0.3785 × log K_{ow} − 2.258, r^2 = 0.4243, p = 0.006; a depuration period of 188 days, log liver/adipose ratios = −0.386 × log K_{ow} + 3.0663, r^2 = 0.4704, $p < 0.001$) (*16*). Similar correlation analyses were also conducted on the basis of the ratios of one wild eagle in the upper peninsula of Michigan (*19*) as shown in Figure 3c, and no statistically significant relationships were obtained, which differed from the results of the present study and laboratory experiments (*16*). The differences may be due to the correlations lacking sufficient representation, as they were based on only one sample data or because the wild eagles in the upper peninsula of Michigan were not in an exposure period.

Recently, Kubota et al. (*17*) used liver/muscle concentration ratios to describe hepatic sequestration. In this study, the average liver/muscle concentration ratios of PCDD/Fs and co-PCBs on a lipid weight basis were also calculated as shown in Table 1. The results show that the average liver/muscle concentration ratios for PCDD/Fs were all higher than 1, and those for coplanar PCBs were mostly lower than 1, which were similar with those reported by Kubota et al. (*17*) (Supporting Information, Figure S3). The liver/muscle concentration ratios in Herring gulls increased significantly with an increase in log K_{ow} (log liver/muscle ratios = 0.3169

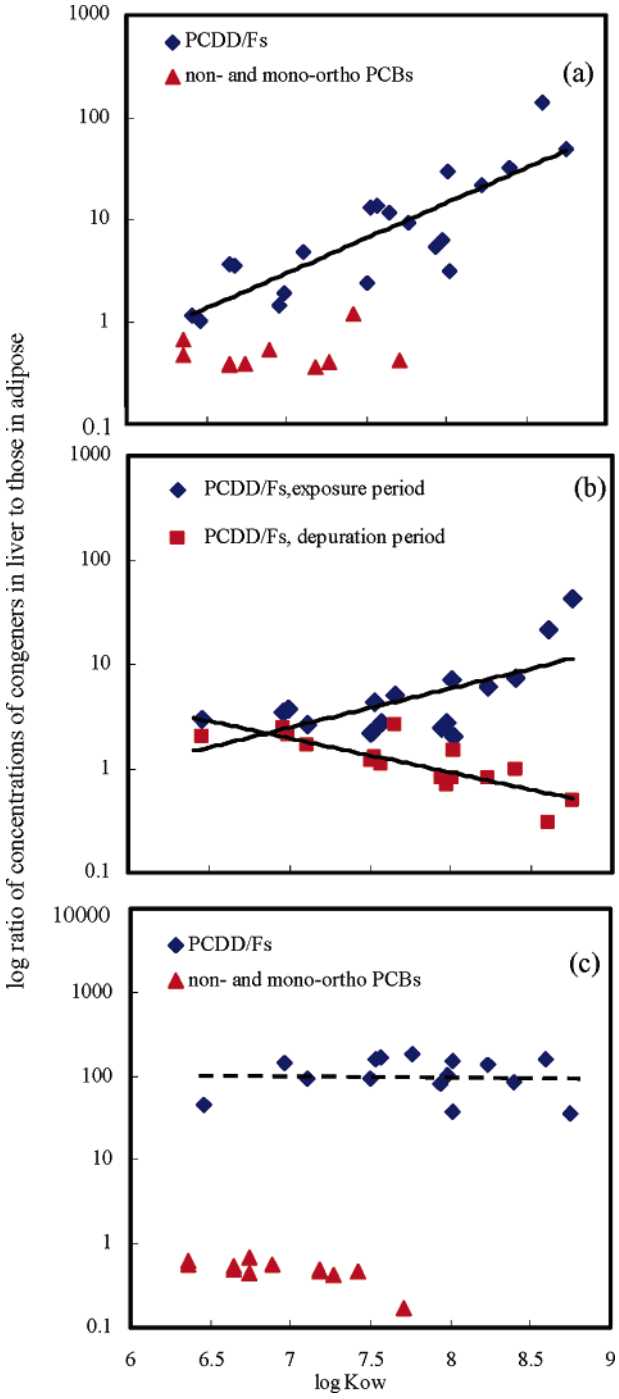

FIGURE 3. Relationships between liver/adipose concentration ratios and log K_{ow} of PCDD/Fs and non- and mono-ortho PCBs in laboratory and wild birds: (a) herring gulls (this study: log liver/adipose ratios = 0.687 × log K_{ow} − 4.3359, r^2 = 0.7058, $p < 0.001$); (b) chicken (*16*) (exposure period of 164 days, log liver/adipose ratios = 0.3785 × log K_{ow} − 2.258, r^2 = 0.4243, p = 0.006; depuration period of 188 days, log liver/adipose ratios = −0.386 × log K_{ow} + 3.0663, r^2 = 0.4704, $p < 0.001$); (c) bald eagles (*19*) (log liver/adipose ratios = −0.0144 × log K_{ow} + 2.107, r^2 = 0.0014, p = 0.893). Dotted line: correlations were not statistically significant. Solid line: correlations were statistically significant. log K_{ow} values were derived on the basis of Govers and Krop (*36*) for PCDD/Fs and Hawker and Connell (*37*) for non- and mono-ortho PCBs.

$\times$ log K_{ow} − 1.7734, $r^2 = 0.2666$, $p = 0.02$); however, the correlation was not statistically significant (log liver/muscle ratios = $-0.0191 \times \log K_{ow} + 0.5162$, $r^2 = 0.0019$, $p = 0.867$) on the basis of Kubota's data (*17*). The differences may be because the cormorants in Lake Biwa were not in an exposure period or due to the species difference of sequestration property.

The mechanism for above hepatic sequestration was originally postulated to be due to the binding of PCDD/Fs to the hepatic microsomal protein (*15*), which was demonstrated by later laboratory researches (*30*, *31*). The significant correlation between liver/adipose ratios for PCDD/Fs and their log K_{ow} values in laboratory chickens in exposure period as well as the wild herring gulls suggested that the binding of PCDD/Fs to the hepatic protein would be dependent on their log K_{ow} values. This study shows the relationships between properties of PCDD/Fs and the degree of the hepatic sequestration in wild birds, which provides structure−activity relationships for estimating future TEF values, since the hepatic sequestration has been shown to be an important pharmacokinetic property in determining TEF values (*7*).

To our knowledge, the liver/adipose ratios for coplanar PCBs in wild birds can only be obtained in reports of Kumar et al. (*19*). In this study, the liver/adipose ratios for coplanar PCBs were less variant (Figure 3a), which was similar to the results of investigations by Kumar et al. (*19*) (Figure 3c), suggesting that deposition of coplanar PCBs, unlike that of PCDD/Fs, would be controlled principally by transport and equilibration among lipid pools of different tissues. Correlation analyses between liver/adipose ratios for coplanar PCBs and log K_{ow} were conducted on the basis of the present data and reference data (*19*), and no significant increasing or decreasing trend was found (Figure 3a,c), indicating that no obvious distribution differences exist between coplanar PCB isomers.

Acknowledgments

Financial support by the Japanese International Cooperation Agency and the National Natural Science Foundation of China (Grant 40021101) is gratefully acknowledged.

Supporting Information Available

Instrumental conditions of dioxin analysis, quantitation and quality assurance quality control, lipid content analysis, identification of the age of herring gulls collected from Bohai Bay, relative contribution of coplanar PCB congeners to the total coplanar PCB concentrations in different tissues of herring gulls, and relationships between liver/muscle concentration ratios and log K_{ow} values of PCDD/Fs and non-, and mono-ortho PCBs in wild birds. This material is available free of charge via the Internet at http://pubs.acs.org.

Literature Cited

(1) Broman, D.; Näf, C.; Rolff, C.; Zebühr, Y.; Fry, B.; Hobbie, J. Using ratios of stable nitrogen isotopes to estimate bioaccumulation and flux of polychlorinated dibenzo-*p*-dixoins (PCDDs) and dibenzofurans (PCDFs) in two food chains from the Northern Baltic. *Environ. Toxicol. Chem.* **1992**, *11*, 331−345.

(2) Naito, W.; Jin, J.; Kang, Y. S.; Yamamuro, M.; Masunaga, S.; Nakanishi, J. Dynamics of PCDD/DFs and coplanar-PCBs in an aquatic food chain of Tokyo Bay. *Chemosphere* **2003**, *53*, 347−362.

(3) Wan, Y.; Hu, J. Y.; Yang, M.; An, L. H.; An, W.; Jin, X. H.; Hattori, T.; Itoh, M. Characterization of Trophic Transfer for Polychlorinated Dibenzo-*p*-dioxins, Dibenzofurans, non- and mono-ortho Polychlorinated Biphenyls in the Marine Food Web of Bohai Bay, North China. *Envrion. Sci. Technol.* **2005**, *39*, 2417−2425.

(4) Safe, S. Polychlorinated biphenyls (PCBs), dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs) and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). *Crit. Rev. Toxicol.* **1990**, *21*, 51−58.

(5) Geyer, H. J.; Schramm, K. W.; Feicht, E. A.; Behechti, A.; Steinberg, C.; Brüggemann, R.; Poiger, H.; Henkelmann, B.; Kettrup, A. Half-lives of tetra-, penta-, hexa-, hepta-, and octachlorodibenzo-*p*-dioxin in rats, monkeys, and humans-a critical review. *Chemosphere* **2002**, *48*, 631−644.

(6) Iwata, H.; Watanabe, M.; Okajima, Y.; Tanabe, S.; Amano, M.; Miyazaki, N.; Petrov, E. A. Toxicokinetics of PCDD, PCDF and coplanar PCB congeners in Baikal seals, *Pusa sibirica*: age-related accumulation, maternal transfer, and hepatic sequestration. *Envrion. Sci. Technol.* **2004**, *38*, 3505−3513.

(7) Van den Berg, M.; Birnbaum, L.; Bosveld, A. T. C.; Vrunström, B.; Cook, P.; Feeley, M.; Giesy, J. P.; Hanberg, A.; Hasegawa, R.; Kennedy, S. W.; Kubiak, T.; Larsen, J. C.; Rolaf van Leeuwen, F. X.; Djien Liem, A. K.; Nolt, C.; Peterson, R. E.; Poellinger, L.; Safe, S.; Schrenk, D.; Tillitt, D.; Tysklind, M.; Younes, M.; Waern, F.; Zacharewski, T. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environ. Health Persp.* **1998**, *106*, 775−792.

(8) DeVito, M. J.; Diliberto, J. J.; Ross, D. G.; Menache, M. G.; Birnbaum, L. S. Dose−Response Relationships for Polyhalogenated Dioxins and Dibenzofurans Following Subchronic Treatment in Mice I. CYP1A1 and CYP1A2 Enzyme Activity in Liver, Lung, and Skin. *Toxicol. Appl. Pharmacol.* **1997**, *147*, 267−280.

(9) Couture, L. A.; Elwell, M. R.; Birnbaum, L. S. Dioxin-like effects Observed in Male Rats Following Exposure to Octachlorodibenzodioxin (OCDD) during 13-week Toxicity Study. *Toxicol. Appl. Pharmacol.* **1988**, *93*, 31−46.

(10) Birnbaum, L. S.; Couture, L. A.; Octachlorodibenzodioxin (OCDD) in Male Rats. *Toxicol. Appl. Pharmacol.* **1988**, *93*, 22−30.

(11) DeVito, M. J.; Birnbaum, L. S. The importance of pharmacokinetics in determining the relative potency of 2,3,7,8-tetrachlorodibenzopdioxin and 2,3,7,8-tetrachlorodibenzofuran. *Fundam. Appl. Toxicol.* **1995**, *24*, 145−148.

(12) Poet, T. S.; Kousba, A. A.; Dennison, S. L.; Timchalk, C. Physiologically Based Pharmacokinetic/Pharmacodynamic Model for the Organophosphorus Pesticide Diazinon. *Neurotoxicology* **2004**, *25*, 1013−1030.

(13) Maruyama, W.; Yoshida, K.; Tanaka, T. Nakanishi, J. Determination of tissue-blood partition coefficients for a physiological model for humans, and estimation of dioxin concentration in tissues. *Chemosphere* **2002**, *46*, 975−985.

(14) Van den Berg, M.; De Jongh, J.; Poiger, H.; Olson, J. R. The Toxicokinetics and Metabolism of Polychlorinated Dibenzo-*p*-Dioxin (PCDDs) and Dibenzofurans (PCDFs) and Their Relevance for Toxicity. *Crit. Rev. Toxicol.* **1994**, *24*, 1−74.

(15) Braune, B. M.; Norstrom, R. J. Dynamics of Organochlorine Compounds in Herring gulls: III. Tissue Distribution and Bioaccumulation in Lake Ontario Gulls. *Environ. Toxicol. Chem.* **1989**, *8*, 957−968.

(16) Stephens R. D., Petreas M. X., Hayward D. G. Biotransfer and bioaccumulation of dioxins and furans from soil: chickens as a model for foraging animals. *Sci. Total Environ.* **1995**, *175*, 253−273.

(17) Kubota, A.; Iwata, H.; Tanabe, S.; Yoneda, K.; Tobata, S. Levels and Toxicokinetic Behaviors of PCDD, PCDFs, and Coplanar PCB Congeners in Common Cormorants from Lake Biwa, Japan. *Environ. Sci. Technol.* **2004**, *38*, 3853−3859.

(18) Wu, W. Z.; Zhang, Q. H.; Schramm, K. W.; Xu, Y.; Kettrup, A. Distribution, Transformation, and Long-Term accumulation of Polychlorinated Dibenzo-*p*-dioxins and Dibenzofurans in Different Tissues of Fish and Piscivorous Birds. *Ecotoxicol. Environ. Saf.* **2000**, *46*, 252−257.

(19) Kumar, K. S.; Kannan, K.; Giesy, J. P.; Masunaga, S. Distribution and elimination of polychlorinated dibenzo-*p*-dioxins, dibenzofurans, biphenyls, and p,p′-DDE in tissue of Bald eagles from the upper Peninsula of Michigan. *Envrion. Sci. Technol.* **2002**, *36*, 2789−2796.

(20) Wan, Y.; Hu, J. Y.; An, L. H.; An, W.; Yang, M.; Itoh, M.; Hattori, T.; Tao, S.; Determination of trophic relationships within a Bohai Bay food web using stable δ^{15}N and δ^{13}C analysis. *Chin. Sci. Bull.* **2005**, *50*, 708−712.

(21) Birds & Birding: A personal electronic journal by Victor W. Fazio III. http://www.aves.net/photoindex/Laridae/Herring_Gull/PH_Herring_Gull_adw-to-summer_25Jan05.html.

(22) Zheng, G. M. *Ornithology*; Beijing Normal University Press: Beijing, 1995; p 20.

(23) Poor, H. H., Plumage and Soft-part Variations in the Herring Gull. *Auk* **1946**, *63* (2), 135−151.

(24) Xiao, F., Zheng, G. M. *Illustrated handbook for common birds*; China Forestry Publishing House: Beijing, 2002; p 143.
(25) Ishaq, R.; Karlson, K.; Näf, C. Tissue distribution of polychlorinated naphthalenes (PCNs) and non-ortho chlorinated biphenyls (non-ortho CBs) in harbour porpoises (*Phocoena phocoena*) from Swedish waters. *Chemosphere* **2000**, *41*, 1913−1925.
(26) Kawai, S.; Fukushima, M.; Miyazaki, N.; Tatsukawa, R. Relationship between lipid composition and organochlorine levels in the tissues of stripped dolphin. *Mar. Pollut. Bull.* **1988**, *19*, 129−133.
(27) Tilbury, K. L.; Stein, J. E.; Meador, J. P.; Krone, C. A.; Chan, S. L. Chemical contaminatants in harbour porpoise (*Phocoena phocoena*) from the north Atlantic coast: tissue concentrations and intra- and inter-organ distribution. *Chemosphere* **1997**, *34*, 2159−2181.
(28) Schneider, R. Polychlorinated biphenyls (PCBs) in cod tissues from the western Baltic: significance of equilibrium partitioning and lipid composition in the bioaccumulation of lipophlilic pollutants in gill-breathing animals. *Meeresforshung* **1982**, *29*, 69−79.
(29) Hu, J. Y.; Wan, Y.; Shao, B.; Jin, X. H.; An, W.; Jin, F.; Yang, M.; Wang, X. J.; Sugisaki, G. Occurrence of Trace Organic Contaminants in Bohai Bay and Its Adjacent Nanpaiwu River, North China. *Mar. Chem.* **2005**, *95*, 1−13.
(30) Diliberto, J. J.; Burgin, D.; Birnbaum, L. S. Role of CYP1A2 in Hepatic Sequestration of Dioxin Studies Using CYP1A2 Knockout Mice. *Biochem. Biophys. Res. Commun.* **1997**, *236*, 431−433.
(31) Diliberto, J. J.; Burgin, D. E.; Birnbaum, L. S. Effects of CYP1A2 on Disposition of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin, 2,3,7,8-Pentachlorodibenzofuran, and 2,2′,4,4′,5,5′-Hexachlorobiphenyl in CYP1A2 Knockout and Parental (C57BL/6N and 129/Sv) Strains of Mice. *Toxicol. Appl. Pharmacol.* **1999**, *159*, 52−64.
(32) Senthil Kumar, K.; Kannan, K.; Paramasivan, O. N.; Shanmuga Sundaram, V. P.; Nakanishi, J.; Masunaga, S. Polychlorinated dibenzo-*p*-dioxins, dienzofurans, and polychlorinated biphenyls in human tissues, meat, fish, and wildlife samples from India. *Environ. Sci. Technol.* **2001**, *35*, 3448−3455.
(33) Tanabe, S.; Watanabe, M.; Minh, T. B.; Kunisue, T.; Nakanishi, S.; Ono, H.; Tanaka, H. PCDDs, PCDFs and coplanar PCBs in albatross from the North Pacific and Southern Oceans: levels, patterns and toxicological implications. *Envrion. Sci. Technol.* **2004**, *38*, 403−413.
(34) Carrier, G.; Brunet, R. C.; Brodeur, J. Modeling of the toxicokinetics of Polychlorinated Dibenzo-*p*-dioxins and Dibenzofurans in mammalians, including humans I. Nonlinear distribution of PCDD/PCDF body burden between liver and adipose tissues. *Toxicol. Appl. Pharmacol.* **1995**, *131*, 253−266.
(35) Carrier, G.; Brunet, R. C.; Brodeur, J.; Modeling of the toxicokinetics of Polychlorinated Dibenzo-*p*-dioxins and Dibenzofurans in mammalians, including humans II. Kinetics of absorption and disposition of PCDDs/PCDFs. *Toxicol. Appl. Pharmacol.* **1995**, *131*, 267−276.
(36) Govers, H. A. J.; Krop, H. B. Partition constants of chlorinated dibenzofurans and dibenzo-*p*-dioxins. *Chemosphere* **1998**, *37* (9−12), 2139−2152.
(37) Hawker, D. W.; Connell, D. W. Octanol−water partition coefficients of polychlorinated biphenyl congeners. *Environ. Sci. Technol.* **1988**, *22*, 382−387.

Received for review November 9, 2005. Revised manuscript received December 19, 2005. Accepted December 21, 2005.

ES052249S