CONTAMINATION STATUS AND ACCUMULATION FEATURES OF DIOXINS AND RELATED COMPOUNDS IN TERRESTRIAL MAMMALS FROM JAPAN

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Abstract

The present study elucidated the contamination status and accumulation features of dioxins and related compounds (DRCs) in the liver of terrestrial mammals such as wild boar (*Sus scrofa*), mongoose (*Herpestes javanicus*), cat (*Felis silvestris catus*) and raccoon (*Procyon lotor*) collected in Japan during 1999-2006. DRCs were detected in all the samples analyzed in this study. TEQ levels were in the order of raccoon > wild boar > cat > mongoose and TEQs in raccoons were one order of magnitude higher than those in other species. These variations between species could be due to food habit, region-specific exposure to DRCs, and/or species-specific accumulation properties such as metabolism and hepatic sequestration of DRCs. When compositions of TEQs in these terrestrial mammals were examined, higher contribution of PCDD/DFs than coplanar-PCBs was observed in all species, suggesting greater dioxin-like risk by PCDD/DFs. DRC congener profiles demonstrated extremely higher contribution of OCDD (> 80%) in all species, especially wild boar and raccoon. This suggests that Japanese wild terrestrial mammals have been chronically exposed to elevated levels of OCDD, perhaps via soil intake.

Introduction

Dioxins and related compounds (DRCs), such as polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and coplanar polychlorinated biphenyls (Co-PCBs), have been found in various wildlife species and humans because of their persistency in the environment and highly bioaccumulative feature, and hence, their toxic effects on humans and wildlife are of great concern.[1] It is suggested that terrestrial and coastal animals, which inhabit regions close to dioxin sources, have been exposed to higher levels of PCDD/DFs than species inhabiting open sea due to their lower transportability than coplanar PCBs.[2]

In Japan, large amounts of DRCs have been released into the environment.[3] But it is estimated that release into the environment and human exposure to DRCs have generally decreased in recent years because of highly efficient incinerators and strict regulations on production and usage of CNP, PCP and PCBs.[4] However, it is suggested that the large amounts of dioxins released during 1960s-1970s still remain in soil and the input from the secondary source may be continuing.[3] Considering these observations, it can be suspected that terrestrial mammals inhabiting contaminated soil areas in Japan have been exposed to relatively high levels of DRCs, and therefore, these species may be at high risk from these contaminants. In fact, our group reported recently that

DRC levels in the liver of wild raccoon dogs were much higher than those in human liver from Japan.[5]

The present study attempted to elucidate the contamination status and accumulation features of DRCs in terrestrial mammals such as wild boar, common mongoose, cat, and raccoon collected from different locations in Japan.

Materials and methods

Liver samples of wild boar (*Sus scrofa*) (*n*=5) and cat (*Felis silvestris catus*) (*n*=4) were collected from Ehime prefecture during 1999-2003. In addition, liver samples of mongoose (*Herpestes javanicus*) (*n*=9) and raccoon (*Procyon lotor*) (*n*=5) were collected from Okinawa and Hokkaido prefectures, respectively, during 2005. All the samples were stored in the Environmental Specimen Bank (*es*-BANK) for Global Monitoring at Ehime University[6] at -25℃ until analysis. DRCs (PCDDs, PCDFs and coplanar PCBs) were analyzed following the method described previously.[2] Identification and quantification of DRCs was performed using HRGC-HRMS. TEQs were calculated using WHO-TEFs proposed in 1998[1] and 2006.[7] Concentrations of DRCs are expressed on lipid weight basis.

Results and Discussion

DRCs were detected in all the liver samples analyzed in this study (Table. 1). In wild boars and raccoons,

Table 1. Concentrations (pg/g lipid wt.) of dioxins and related compounds in the liver of wild boars, mongooses, cats and raccoons from Japan.

	Wild boar (*n* = 5)		Mongoose (*n* =9)		Cat (*n* =4)		Raccoon (*n* =5)	
	Mean±SD	(Range)	Mean±SD	(Range)	Mean±SD	(Range)	Mean±SD	(Range)
Dioxins								
2,3,7,8-TetraCDD	0.90±0.25	(0.52-1.1)	<0.05		1.6±2.6	(<0.05-5.4)	7.8±5.2	(<0.05-7.8)
1,2,3,7,8-PentaCDD	7.2±4.0	(2.6-13)	0.10±0.33	(<0.05-1.0)	5.0±6.6	(0.63-15)	63±47	(21-63)
1,2,3,4,7,8-HexaCDD	37±14	(19-57)	3.9±2.4	(<0.1-8.3)	18±30	(2.1-64)	50±25	(20-50)
1,2,3,6,7,8-HexaCDD	24±13	(11-40)	1.7±1.5	(<0.1-4.2)	6.7±8.7	(1.4-20)	120±110	(38-120)
1,2,3,7,8,9-HexaCDD	9.1±6.9	(2.4-20)	0.40±0.82	(<0.1-1.9)	12±19	(<0.10-41)	25±16	(9.5-25)
1,2,3,4,6,7,8-HeptaCDD	340±210	(160-680)	54±22	(20-84)	400±670	(38-1400)	1500±480	(920-1500)
OctaCDD	9100±5200	(5300-18000)	7900±6800	(1700-24000)	4500±7700	(440-16000)	44000±17000	(24000-44000)
Furans								
2,3,7,8-TetraCDF	0.30±0.29	(<0.05-0.61)	0.70±0.40	(<0.05-1.2)	0.65±0.44	(<0.05-0.97)	2.8±2.7	(<0.05-2.8)
1,2,3,7,8-PentaCDF	0.033±0.073	(<0.05-0.16)	<0.05		0.80±0.69	(<0.1-1.7)	2.7±4.3	(<0.05-2.7)
2,3,4,7,8-PentaCDF	76±19	(48-91)	12±7.9	(3.4-29)	31±39	(4.9-88)	40±21	(20-40)
1,2,3,4,7,8-HexaCDF	68±24	(43-110)	11±5.6	(4.4-21)	18±22	(3.4-52)	290±81	(220-290)
1,2,3,6,7,8-HexaCDF	56±13	(37-72)	1.3±1.3	(<0.1-3.5)	21±30	(3.3-65)	36±15	(21-36)
1,2,3,7,8,9-HexaCDF	<0.1		<0.1		<0.1		<0.10	
2,3,4,6,7,8-HexaCDF	64±15	(51-86)	2.0±1.1	(<0.1-4.1)	41±53	(8.2-120)	110±120	(36-110)
1,2,3,4,6,7,8-HeptaCDF	220±88	(110-330)	6.1±3.8	(1.5-14)	21±22	(4.4-53)	290±99	(180-290)
1,2,3,4,7,8,9-HeptaCDF	19±6.8	(9.1-28)	<0.1		0.80±1.7	(<0.1-3.3)	63±21	(40-63)
OctaCDF	54±43	(7.5-100)	0.80±1.5	(<0.2-3.9)	8.6±6.5	(2.0-18)	710±340	(260-710)
Non-***ortho PCBs***								
3,3',4,4'-TetraCB (77)	2.2±0.62	(1.7-3.2)	28±38	(4.2-120)	43±33	(14-90)	9.4±4.6	(3.7-9.4)
3,4,4',5-TetraCB (81)	1.0±0.67	(0.36-2.1)	3.2±1.0	(2.1-4.7)	3.7±2.2	(1.5-6.8)	12±6.2	(3.8-12)
3,3',4,4',5-PentaCB (126)	53±38	(18-120)	35±18	(13-70)	38±28	(17-78)	260±110	(95-260)
3,3',4,4',5,5'-HexaCB (169)	98±61	(28-170)	40±39	(14-140)	71±87	(18-200)	77±29	(46-77)
Mono-***ortho PCBs***								
2,3,3',4,4'-PentaCB (105)	1300±2700	(65-6200)	1200±1500	(130-4700)	4700±6200	(1300-14000)	1700±1100	(520-1700)
2,3,4,4',5-PentaCB (114)	110±240	(<2.0-550)	23±47	(<2.0-120)	340±300	(<2.0-720)	120±80	(43-120)
2,3',4,4',5-PentaCB (118)	2700±5600	(120-13000)	8900±12000	(460-37000)	16000±21000	(4200-48000)	4900±2900	(1700-4900)
2',3,4,4',5-PentaCB (123)	32±60	(<2.0-140)	19±37	(<2.0-90)	33±65	(<2.0-130)	4.2±9.4	(<2.0-21)
2,3,3',4,4',5-HexaCB (156)	1100±1500	(220-3700)	1200±1900	(200-6200)	3100±4600	(720-10000)	1600±1100	(470-1600)
2,3,3',4,4',5'-HexaCB (157)	370±340	(150-930)	200±170	(48-590)	1100±1500	(280-3400)	300±210	(97-300)
2,3',4,4',5,5'-HexaCB (167)	290±530	(40-1200)	2700±4700	(270-15000)	860±960	(320-2300)	340±190	(130-340)
2,3,3',4,4',5,5'-HeptaCB (189	260±150	(110-430)	2800±5200	(190-16000)	610±790	(190-1800)	300±250	(88-300)
TEQs (1998TEF)	86±26	(58-120)	15±6.7	(6.0-26)	47±57	(11-130)	210±100	(130-370)
TEQs (2006TEF)	74±23	(50-100)	14±5.0	(4.6-20)	40±45	(9.4-110)	210±95	(130-360)

SD; standard deviation

PCDDs (wild boar: 9,500 ± 5,400 pg/g; raccoon: 45,000 ± 17,000 pg/g) were dominant, followed by mono-*ortho* PCBs (wild boar: 6,200 ± 11,000 pg/g; raccoon: 9,200 ± 5,800 pg/g), PCDFs (wild boar: 560 ± 160 pg/g; raccoon: 1,500 ± 350 pg/g) and non-*ortho* PCBs (wild boar: 150 ± 67 pg/g; raccoon: 360 ± 140 pg/g). Concentrations of DRCs in mongooses and cats were in the order of mono-*ortho* PCBs (mongoose: 17,000 ± 25,000 pg/g; cat: 27,000 ± 36,000 pg/g) > PCDDs (mongoose: 8,000 ± 6,800pg/g; cat: 4,900 ± 7,600 pg/g) > non-*ortho* PCBs (mongoose: 110 ± 46 pg/g; cat: 160 ± 110 pg/g) > PCDFs (mongoose: 34 ± 17 pg/g; 140 ± 170 pg/g). Accumulation levels of PCDD/DFs in raccoons were one order of magnitude higher than those in other species, whereas mono-*ortho* PCBs in cat and mongoose were relatively higher than those in other two species. These variations between species could be due to food habit, region-specific exposure and/or species specific metabolic capacity for DRCs. In addition, high accumulation levels of PCDD/DFs in the liver of raccoons might be caused by hepatic sequestration of these compounds, as reported in raccoon dog,[5] which belong to the canine family.

TEQs in terrestrial mammals were almost similar between the values calculated using 1998-TEFs[1] and 2006-TEFs[7] (Table 1). When compositions of TEQs were examined, higher contribution of PCDD/DFs than co-PCBs was observed in all species (Figure 1), suggesting greater dioxin-like risk by PCDD/DFs, especially for raccoon and raccoon dog because of elevated levels of these contaminants.

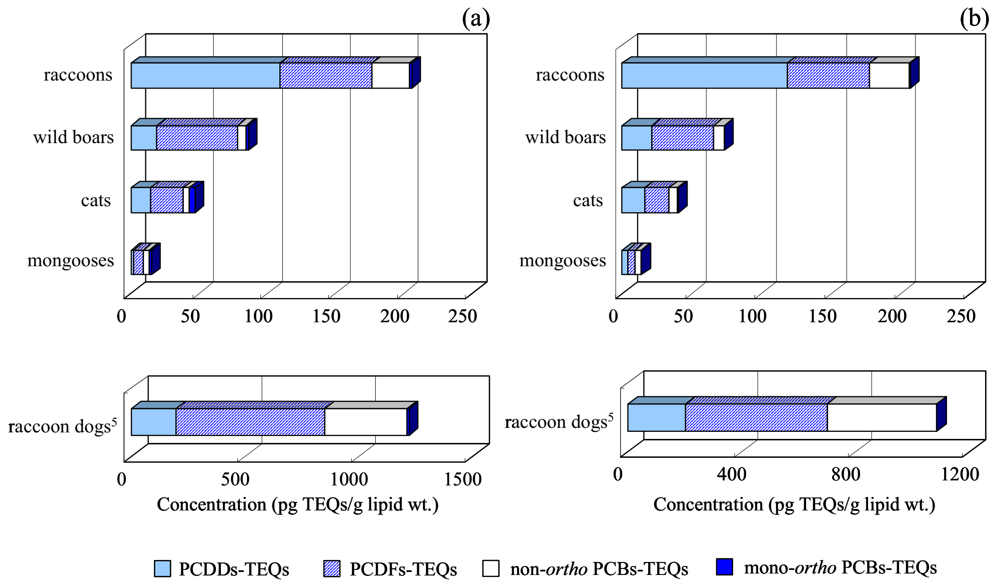

Figure 1. Comparison of TEQ levels in wild terrestrial mammals analyzed in this study with those in raccoon dogs reported previously. TEQ values were estimated based on TEFs proposed by the WHO in 1998 (a) and 2006 (b). (5) reference

Among PCDD/DF congeners, OCDD was dominant in all the species, followed by 1,2,3,4,6,7,8-HpCDD (Table 1). Such an accumulation pattern was also noticed in raccoon dogs reported previously.[5] On the other hand, less contribution of OCDD was observed in common cormorants from Lake Biwa,[8] which is a fish-eating avian species. In a study using experimental animals,[9] it has been reported that gastrointestinal absorption of OCDD was extremely poor. These observations suggest that Japanese wild terrestrial mammals have been chronically exposed to elevated levels of OCDD. Pentachlorophenol (PCP), which contained OCDD as impurities, was abundantly used in agricultural fields during the 1960s and 1970s in Japan, and recently relatively high concentration of OCDD derived from PCP were detected in soils.[3] Hence, wild terrestrial mammals may have been exposed to DRCs via soil in addition to their food intake.

Acknowledgements

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References

1) Van den Berg M, Brinbaum L, Bosveld ATC,Brunstrom B, Cook P, Feeley M, Giesy JP, Hanberg A, Hasegawa R, Kennedy SW, Kubiak T, Larsen JC, Rolaf van Leeuwen FX, Liem AKD, Nolt C, Peterson RE, Poellinger L, Safe S, Schrenk D, Tillit D, Tysklind M, Younes M, Waern F, Zacharewski T. *Environ Health Perspect* 1998; 106; 775-792.
2) Tanabe S, Watanabe M, Minh TB, Kunisue T, Nakanishi S, Ono H, Tanaka H. *Environ Sci Technol* 2004; 38; 403-413.
3) Masunaga S, Yao Y, Ogura I, Sakurai T, Nakanishi J. *Chemosphere* 2003; 53; 315-324.
4) Yoshida K, Nakanishi J. *Chemosphere* 2003; 53; 427-436.
5) Kunisue T, Watanabe XM, Iwata H, Tsubota T, Yamada F, Yasuda M, Tanabe S. *Environ Poll* 2006; 140; 525-535.
6) Tanabe S. *J Environ Monit* 2006 ; 8 ; 782-790.
7) Van den Berg M, Birnbaum L, Denison M, De Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Tritscher A, Tuomisto J, Tysklind M, Walker N, Peterson RE. *Toxicol Sci* 2006; 93(2); 223-241.
8) Kubota A, Iwata H, Tanabe S, Yoneda K, Tobata S. *Environ Sci Technol* 2004; 38; 3853-3859.
9) Birnbaum LS, Couture LA. *Toxicol Appl Pharmacol* 1988; 93; 22-30.