Half-life of each dioxin and PCB congener in the human body

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Introduction

It is well known that dioxin and PCB congeners accumulate in the human body. For assessing their toxicological risk, it is important to know the half-life of each congener in the human body.

This study summarizes the overall half-lives of congeners in humans as reported in the literature, and compares them with the half-lives due to fecal and sebum excretions, as estimated by data on the concentrations of congeners in feces and sebum in the literature. In addition, the overall half-lives of congeners for the general Japanese population were estimated from the data on dietary intakes and concentrations in the human body reported by the municipalities.

Overall half-lives of dioxins and PCBs in the human body

Available data on the elimination half-lives of congeners[1-10] are summarized in Table 1. The half-lives for most congeners ranged from a few years to approximately 20 years. All experimental data were derived from subjects who had been accidentally exposed to high levels of dioxins and/or PCBs[1-8], while the half-lives for the general population who had only been exposed to background levels of these substances were estimated by a one-compartment kinetic model with first-order elimination from lipids[9,10].

There is little available data on the elimination half-lives of dioxin-like PCBs (WHO-TEF (Toxic Equivalent Factor) > 0), despite the fact that such PCBs make up more than 50% of the total TEQ (toxic equivalent) of human intake in Japan[11].

Elimination of dioxins and PCBs by fecal excretion

Rohde et al.[6] have reported the half-lives of dioxins due to fecal excretion in addition to the overall half-lives. In addition, several studies[12-15] have reported both the daily excretion of congeners via feces (E_f [pg/day]) and the concentrations

of congeners in blood lipids (C_b [pg/g-lipid]). Based on those data, and assuming that the concentrations of congeners in blood lipids are appropriate surrogates for the concentrations in total lipids; that all congeners are sequestered in lipids; and that total body lipid weight is 12000 g, we calculated the half-lives due to fecal excretion ($T_{f1/2}$ [year]) as

$$T_{f1/2} = \ln (2) / (E_f / C_b / 12000) / 365 \quad (1)$$

The half-lives due to fecal excretion obtained by the data from different studies (Table 2) were similar. Although the obtained half-lives due to fecal excretion were generally longer than the overall half-lives (Table 1), fecal excretion seems to be responsible for a large part of the overall elimination of congeners.

It has been reported that the concentrations of congeners in bile lipids are the same level as those in blood lipids[16]. The measured excretion of congeners via feces is considered to include in part the excretion of congeners via bile, because bile is excreted in the intestines.

Elimination of dioxins and PCBs by sebum excretion

Some studies[15,17,18] have reported the concentrations of congeners in sebum (C_s [pg/g-lipid]) in addition to those in blood lipids. Based on those data, and assuming that the concentrations of congeners in blood lipids are appropriate surrogates for the concentrations in total lipids; that all congeners are sequestered in lipids; that daily sebum secretion is 0.75 g-lipid/day; and that total body lipid weight is 12000 g, we calculated the half-lives due to sebum excretion ($T_{s1/2}$ [year]) as

$$T_{s1/2} = \ln (2) / (C_s \times 0.75 / C_b / 12000) / 365 \quad (2)$$

The obtained half-lives due to sebum excretion (Table 3) were generally longer than the obtained half-lives due to fecal excretion (Table 2), except in the case of certain congeners, such as PCB-77.

Overall half-lives of dioxins and PCBs for the general Japanese population as estimated from recent data

Dioxins and PCBs mostly enter the human body via food. The overall half-lives ($T_{1/2}$ [year]) of congeners in the human body under steady-state conditions—assuming that the concentrations of congeners in the lipids of blood, adipose tissue, or breast milk are appropriate surrogates for the concentrations in total lipids; that all congeners are sequestered in lipids; and that total body lipid weight is 12000 g—can be calculated from the recent data on the dietary intakes of congeners[11] and the concentrations of congeners in the human body[19-21] as

$$T_{1/2} = C_{lipid} \times 12000 \times \ln (2) / (Abs \times I \times 365) \quad (3)$$

where C_{lipid} [pg/g-lipid] is the concentration of a congener in body lipids, *Abs* [-] is gastrointestinal absorption[13] (see Table 4), and I [pg/day] is the dietary intake of a congener.

The obtained half-lives assuming steady-state conditions (Table 4) may be biased, especially for congeners having a longer half-life (ex. >10), because of historical decreases in the average intake level of dioxins and PCBs[11]. However, the obtained half-lives for congeners having a shorter half-life and the relative lengths of half-lives among congeners could be fairly reliable. The estimated half-lives (Table 4) are consistent with those in the literature (Table 1). Although there is some evidence that at high exposures liver enzymes that increase the elimination rate of congeners are induced[25], the differences in the elimination rates between individuals subjected to background exposures (Table 4) and those subjected to high exposures (Table 1) are unclear.

Overall half-lives of dioxins and PCBs for the general Japanese population as estimated from longitudinal exposure data

The overall half-lives derived from body burden due to cumulative exposure from food were estimated by a one-compartment kinetic model with first-order elimination from lipids, assuming that the concentrations of congeners in breast milk lipids are appropriate surrogates for the concentrations in total lipids and that all congeners are sequestered in lipids, according to the formulae[26]

$$d\{W_{lipid} \times C_{lipid}\} / dt = Abs \times \sum_i \{I_{food,i} \times C_{food,i}\} - k \times C_{lipid} \qquad (4)$$

$$T_{1/2} = \ln(2) \times W_{lipid} / k / 365 \qquad (5)$$

where W_{lipid} [g-lipid] is the total body lipid weight, $I_{food,i}$ [g/day] is the dietary food intake of food group i, $C_{food,i}$ [pg/g] is the concentration of a congener in food of food grourp i, and k [g-lipid/day] is the first-order elimination rate from lipids.

In the model, the dietary food intake ($I_{food,i}$) and total body lipids (W_{lipid}) were functions of age, the year, and sex[27]. Because Japanese are exposed to dioxins and PCBs mostly through fish, meat, dairy products, and colored vegetables[11], only these four food groups were considered in the model. The concentrations of congeners in food ($C_{food,i}$) in each year were estimated from total diet study samples from the Kansai region in Japan collected in FY1977-FY2000[11] and other information such as the time trends of the concentrations of total PCBs in food[28,29] and the time trends of environmental concentrations[30,31]. The concentrations of congeners in breast milk from Osaka in the Kansai region[19] were used as estimates of the body burden. Therefore, the target population was women who were 25-34 years old in 1998.

The value of k for each congener was determined by the calculating concentration to fit the observed concentration in breast milk in subjects aged 25-34 years in 1998 under the following three conditions: (1) k is constant irrespective of age; (2) k is proportional to daily food intake, which is a function of age; (3) k is proportional to $(W_{liver})^{2/3}$, where W_{liver} is liver weight, which is a function of age[26]. There were few differences of estimated half-lives at the age of 25-34 among the three conditions, although the estimated half-lives in childhood were different among the three conditions.

The obtained half-lives for individuals aged 25-34 in 1998 under the condition (1) are shown in the far right column of Table 4 (the average of W_{lipid} for individuals aged 25-34 in 1998 was 12400 g[27]). The differences between the half-lives estimated under unsteady-state conditions and those estimated under steady-state conditions were not so large. The estimated half-life of PCB-126, which is the largest contributor to the TEQ of human intake in Japan[11], was approximately 4.5 years.

Table 1. Overall half-lives ($T_{1/2}$) of dioxins and PCBs in the human body [year].

2,3,7,8-T_4CDD	7.2					8.5	8.7	8.2	6.2	7.8	6.3
1,2,3,7,8-P_5CDD	15.7					14			8.6	11	8.3
1,2,3,4,7,8-H_6CDD	8.4					14			19	12	7.8
1,2,3,6,7,8-H_6CDD	13.1	3.5				8.5			>70	12	10
1,2,3,7,8,9-H_6CDD	4.9					7.1			8.5	6.8	4.6
1,2,3,4,6,7,8-H_7CDD	3.7	3.2				4.3			6.6	8.8	3.2
O_8CDD	6.7	5.7				9			5.6	5.7	4.6
2,3,7,8-T_4CDF									0.4	1.4	2.4
1,2,3,7,8-P_5CDF									0.9	2.9	3.9
2,3,4,7,8-P_5CDF	19.6		4.5	3.1	8.9	14			9.9	10	7.8
1,2,3,4,7,8-H_6CDF	6.2		4.0	3.3	5.4	8.3			5.7	7.7	5.6
1,2,3,6,7,8-H_6CDF	6		4.9			7.6			6.2	24	7.1
1,2,3,7,8,9-H_6CDF											
2,3,4,6,7,8-H_6CDF	5.8					11			2.4	3.6	3.1
1,2,3,4,6,7,8-H_7CDF	3	<1.7	6.8	2.4	3.9	3.6			2.6	5.0	2.8
1,2,3,4,7,8,9-H_7CDF	3.2									10	5.2
O_8CDF		1.8							<0.2	0.7	1.6
PCB-77									0.1		
PCB-118				1.7	17.6						
PCB-126									2.7		
PCB-138				4.8	15.2						
PCB-153				3.9	10.6						
PCB-156				4.9	13.4						
PCB-169				about 10					13		
PCB-170				5.5	14.7						
PCB-180				5.4	14.9						
footnote	*1	*2	*3	*4	*5	*6	*7	*8	*9	*10	*11

*1: German herbicide plant workers[1] (*n* =5-48). Half-lives were calculated from decay of the concentrations in blood from 1985/86 to 1992/94.

*2: A person exposed to technical pentachlorophenol in wood wall of home[2] (*n* =1). Half-lives were calculated from decay of the concentrations in adipose tissue for 28 months.

*3: An American male worker at the Binghamton state office building where a PCB transformer fire occurred[3] (*n* =1). Half-lives were calculated from decay of the concentrations in blood and adipose tissue for 6 years.

*4: Yu-Cheng patients[4,5] (*n* =3). Half-lives were calculated from decay of the concentrations in blood taken from 0.6 to 15.6 years subsequent to first exposure.

*5: Yusho patients[4,5] (*n* =5). Half-lives were calculated from decay of the concentrations in blood taken from 14 to 29.1 years subsequent to first exposure.

*6: Male chemical-plant workers[6] (*n* =6). Half-lives were calculated from decay of the concentrations in blood from 1990/92 to 1996 (values taken from figures).

*7: Ranch Hand veterans[7] (*n* =213). Half-lives were calculated from decay of the concentrations in blood from 1982 to 1992.

*8: Residents in Seveso, Italy[8] (*n* =27). Half-lives were calculated from decay of the concentrations in blood.

*9: The general Dutch population[9]. Half-lives derived from body burden after 28 years of cumulative exposure from food were estimated by a one-compartment kinetic model with first-order elimination from lipids.

*10: The general Dutch population[10] (background). Half-lives at 48.7 years of age were estimated from cross-sectional data using a one-compartment kinetic model with first-order elimination from lipids.

*11: German herbicide plant workers[10]. Half-lives at 48.7 years of age were estimated from longitudinal data[1] using a one-compartment kinetic model with first-order elimination from lipids.

Table 2. Daily excretion of dioxins and PCBs via feces (E_f) per concentration in blood (C_b) and estimated half-lives in humans due to fecal excretion ($T_{f\,1/2}$).

congeners	n	E_f/C_b [(pg/day)/(pg/g-lipid)]	$T_{f\,1/2}$ [year]	n	E_f/C_b [(pg/day)/(pg/g-lipid)]	$T_{f\,1/2}$ [year]	n	E_f/C_b [(pg/day)/(pg/g-lipid)]	$T_{f\,1/2}$ [year]	n	E_f/C_b [(pg/day)/(pg/g-lipid)]	$T_{f\,1/2}$ [year]	n	E_f/C_b [(pg/day)/(pg/g-lipid)]	$T_{f\,1/2}$ [year]
2,3,7,8-T_4CDD	6	1.82	**22**	7	1.06	**22**	5	1.16	**20**	17	0.87	**22**	3	0.89	**25**
1,2,3,7,8-P_5CDD	6	1.58	**28**	7	1.13	**20**	5	1.26	**18**	17	1.50	**13**	3	1.46	**16**
1,2,3,4,7,8-H_6CDD	6	1.92	**22**	7	1.31	**17**	5	1.68	**14**				3	1.19	**19**
1,2,3,6,7,8-H_6CDD	6	1.80	**22**	7	1.29	**18**	5	1.61	**14**	17	1.36	**14**	3	0.94	**24**
1,2,3,7,8,9-H_6CDD	6	3.54	**12**	7	2.62	**9**	5	2.42	**9.4**				3	1.39	**16**
1,2,3,4,6,7,8-H_7CDD	6	3.10	**15**	7	2.46	**9**	5	2.63	**8.7**	17	1.68	**11**	3	2.00	**11**
O_8CDD	6	4.45	**10**	7	4.26	**5**	5	4.13	**5.5**	17	3.10	**6.1**	3	1.22	**19**
2,3,7,8-T_4CDF										17	1.37	**14**	3	0.67	**34**
1,2,3,7,8-P_5CDF													2	1.60	**14**
2,3,4,7,8-P_5CDF	6	1.56	**33**	7	1.06	**22**	5	1.33	**17**	17	1.51	**13**	3	1.03	**22**
1,2,3,4,7,8-H_6CDF	6	2.16	**20**	7	1.64	**14**	5	2.35	**9.7**	17	1.59	**12**	3	1.39	**16**
1,2,3,6,7,8-H_6CDF	6	2.10	**23**	7	1.41	**16**	5	1.89	**12**				3	1.65	**14**
1,2,3,7,8,9-H_6CDF													1	1.33	**17**
2,3,4,6,7,8-H_6CDF	6	2.58	**25**	4	1.41	**16**	2	1.30	**18**				3	2.05	**11.1**
1,2,3,4,6,7,8-H_7CDF	6	2.27	**25**	7	2.30	**10**	5	2.73	**8.3**	17	1.65	**11**	3	6.08	**3.7**
1,2,3,4,7,8,9-H_7CDF															
O_8CDF				2	1.88	**12**							2	5.44	**4.2**
PCB-28				7	1.44	**16**									
PCB-52				5	2.20	**10**									
PCB-77										17	4.13	**4.6**	3	2.65	**8.6**
PCB-99							5	0.81	**28**						
PCB-101				7	2.07	**11**									
PCB-105				7	0.67	**34**	3	0.74	**31**						
PCB-118							4	0.81	**28**						
PCB-126				3	0.54	**43**	2	0.53	**43**	17	1.43	**13**	3	0.67	**34**
PCB-138				7	0.85	**27**	5	0.63	**36**						
PCB-153				7	0.78	**29**	5	0.56	**41**						
PCB-169										17	1.54	**12**	3	0.57	**40**
PCB-170/190							5	0.39	**59**						
PCB-180				7	0.34	**67**	5	0.35	**65**						
PCB-202				7	0.39	**58**	5	0.42	**54**						
PCB-209							4	0.35	**65**						
Samples	Male chemical-plant workers[6]. Values were taken from the figures.			German male & female volunteers[12]. Half-lives were calculated by Eq. 1.			German male volunteers[13]. It was assumed that the amount of feces was 35 g-dry/day. Half-lives were calculated by Eq. 1.			Yu-Cheng patients[14]. Half-lives were calculated by Eq. 1.			Healthy Japanese men[15]. Half-lives were calculated by Eq. 1.		

Table 3. Ratio of the conc. in sebum (C_s) to that in blood (C_b) and estimated half-lives due to sebum excretion ($T_{s\,1/2}$).

congeners	n	C_s/C_b [(pg/g-lipid)/(pg/g-lipid)]	$T_{s\,1/2}$ [year]	n	C_s/C_b [(pg/g-lipid)/(pg/g-lipid)]	$T_{s\,1/2}$ [year]	n	C_s/C_b [(pg/g-lipid)/(pg/g-lipid)]	$T_{s\,1/2}$ [year]	n	C_s/C_b [(pg/g-lipid)/(pg/g-lipid)]	$T_{s\,1/2}$ [year]	n	C_s/C_b [(pg/g-lipid)/(pg/g-lipid)]	$T_{s\,1/2}$ [year]
2,3,7,8-T_4CDD	<39	0.62	**49**	<42	0.36	**84**	<31	0.67	**46**	8	0.76	**40**	3	1.2	**26**
1,2,3,7,8-P_5CDD	<39	0.65	**47**	<42	0.57	**54**	<31	0.53	**57**	8	0.63	**48**	3	0.98	**31**
1,2,3,4,7,8-H_6CDD	<39	0.67	**46**	<42	0.57	**53**	<31	0.68	**45**	7	0.66	**46**	3	1.2	**26**
1,2,3,6,7,8-H_6CDD	<39	0.40	**77**	<42	0.50	**61**	<31	0.45	**68**	8	0.60	**51**	3	0.71	**43**
1,2,3,7,8,9-H_6CDD	<39	0.37	**83**	<42	0.38	**81**	<31	0.67	**45**	5	0.63	**48**	3	1.3	**23**
1,2,3,4,6,7,8-H_7CDD	<39	3.52	**8.6**	<42	3.00	**10**	<31	3.64	**8.3**	8	5.0	**6.1**	3	11	**2.7**
O_8CDD	<39	1.91	**16**	<42	2.93	**10**	<31	3.78	**8.0**	8	4.6	**6.5**	3	30	**1.0**
2,3,7,8-T_4CDF	<39	2.93	**10**	<42	1.35	**22**	<31	2.47	**12**	8	2.1	**15**	3	12	**2.5**
1,2,3,7,8-P_5CDF	<39	1.08	**28**	<42	0.98	**31**	<31	2.42	**13**	8	1.9	**16**	2	7.6	**4.0**
2,3,4,7,8-P_5CDF	<39	0.48	**64**	<42	0.48	**63**	<31	0.68	**44**	8	0.65	**47**	3	1.4	**21**
1,2,3,4,7,8-H_6CDF	<39	0.30	**100**	<42	0.27	**113**	<31	0.56	**54**	8	0.62	**49**	3	1.5	**20**
1,2,3,6,7,8-H_6CDF	<39	0.33	**91**	<42	0.17	**179**	<31	0.63	**48**	8	0.97	**31**	3	1.5	**20**
1,2,3,7,8,9-H_6CDF	<39	0.38	**80**	<42	0.33	**92**	<31	2.80	**11**	1	0.64	**47**	3	3.0	**10**
2,3,4,6,7,8-H_6CDF	<39	0.86	**35**	<42	0.98	**31**	<31	1.63	**19**	2	0.88	**35**	1	6.3	**4.8**
1,2,3,4,6,7,8-H_7CDF	<39	3.08	**9.9**	<42	1.37	**22**	<31	2.97	**10**	8	3.5	**8.6**	3	4.7	**6.4**
1,2,3,4,7,8,9-H_7CDF															
O_8CDF													2	12	**2.6**
PCB-77	<39	26.9	**1.1**				<31	13.87	**2.2**	8	32	**0.94**	3	73	**0.42**
PCB-126	<39	1.0	**29**	<42	1.03	**30**	<31	0.98	**31**	8	1.1	**29**	3	1.3	**23**
PCB-169	<39	0.5	**58**	<42	0.69	**44**	<31	0.49	**62**	8	0.5	**64**	3	0.52	**58**
Samples	Yusho patients[17]. Half-lives were calculated by Eq. 2.			Yu-Cheng patients[17]. Half-lives were calculated by Eq. 2.			Normal volunteers[17]. Half-lives were calculated by Eq. 2.			Male volunteers in Fukuoka, Japan[18]. Half-lives were calculated by Eq. 2.			Healthy Japanese men[15]. Half-lives were calculated by Eq. 2.		

Table 4. Estimated overall half-lives ($T_{1/2}$) of dioxins and PCBs for the general Japanese population [year].

Congeners	*Abs**	$T_{1/2}$**		$T_{1/2}$**		$T_{1/2}$**		$T_{1/2}$***	
2,3,7,8-T_4CDD	98%	**11**	(9.5 - 12)			**6.7**	(3.3 - 14)	**6.3**	(4.0 - 11)
1,2,3,7,8-P_5CDD	94%	**11**	(9.5 - 12)	**6.7**	(4.9 - 9.6)	**6.6**	(3.6 - 12)	**11**	(7.3 - 22)
1,2,3,4,7,8-H_6CDD	87%							**8.8**	(5.1 - 18)
1,2,3,6,7,8-H_6CDD	86%	**46**	(41 - 52)	**42**	(29 - 60)	**24**	(12 - 50)	**37**	(14 -)
1,2,3,7,8,9-H_6CDD	85%					**9.2**	(3.2 - 27)		
1,2,3,4,6,7,8-H_7CDD	71%	**4.0**	(3.5 - 4.5)	**5.8**	(4.0 - 8.3)	**1.4**	(0.68 - 3.0)	**5.6**	(3.9 - 8.4)
O_8CDD	53%	**5.5**	(4.7 - 6.4)	**22**	(18 - 26)	**5.0**	(1.8 - 14)	**8.6**	(5.5 - 15)
2,3,7,8-T_4CDF	99%	**0.35**	(0.3 - 0.4)			**0.17**	(0.081 - 0.4)	**0.88**	(0.54 - 1.4)
1,2,3,7,8-P_5CDF	98%					**0.42**	(0.18 - 1.0)	**1.1**	(0.58 - 1.9)
2,3,4,7,8-P_5CDF	97%	**6.7**	5.9 - 7.5)	**4.9**	(3.3 - 7.1)	**5.0**	(2.7 - 9.1)	**9.4**	(5.7 - 19)
1,2,3,4,7,8-H_6CDF	94%	**10**	(8.6 - 12)	**9.9**	(6.6 - 15)	**3.7**	(1.3 - 10)	**7.3**	(4.9 - 12)
1,2,3,6,7,8-H_6CDF	94%	**14**	(12 - 17)	**17**	(11 - 26)	**5.8**	(1.4 - 25)	**8.4**	(5.4 - 15)
1,2,3,7,8,9-H_6CDF	91%								
2,3,4,6,7,8-H_6CDF	93%	**4.8**	(3.8 - 6.1)			**2.1**	(0.8 - 5.8)	**4.8**	(3.0 - 8.4)
1,2,3,4,6,7,8-H_7CDF	86%	**2.2**	(1.9 - 2.7)	**4.8**	(3.2 - 7.2)	**1.4**	(0.5 - 3.8)	**3.9**	(2.6 - 6.2)
1,2,3,4,7,8,9-H_7CDF	79%								
O_8CDF	61%					**2.1**	(0.7 - 6.2)		
PCB-77	99%	**0.34**	(0.28 - 0.40)			**0.070**	(0.046 - 0.11)	**0.74**	(0.38 - 1.3)
PCB-81	99%	**1.2**	(0.91 - 1.5)			**0.73**	(0.4 - 1.2)	**1.7**	(0.96 - 2.8)
PCB-105	98%	**4.0**	(3.4 - 4.8)	**2.4**	(1.7 - 3.3)	**2.7**	(1.5 - 4.8)	**5.2**	(2.5 - 12)
PCB-114	98%	**19**	(16 - 22)	**10**	(7.4 - 14.2)	**25**	(16 - 40)	**15**	(7.4 - 51)
PCB-118	98%	**5.4**	(4.7 - 6.3)	**3.8**	(2.8 - 5.3)	**4.2**	(2.3 - 7.5)	**6.3**	(3.8 - 12)
PCB-123	98%	**4.2**	(3.5 - 5.0)	**7.4**	(5.3 - 10)	**12**	(5.8 - 25)	**4.6**	(2.2 - 11)
PCB-126	98%	**3.0**	(2.6 - 3.3)	**1.6**	(1.2 - 2.1)	**2.7**	(1.6 - 4.5)	**4.5**	(2.7 - 7.8)
PCB-156	95%	**19**	(17 - 23)	**16**	(11 - 23)	**38**	(23 - 63)	**19**	(9.2 - 80)
PCB-157	97%	**18**	(16 - 22)	**18**	(13 - 26)	**27**	(16 - 44)	**12**	(6.0 - 36)
PCB-167	96%	**10**	(8.6 - 12)	**12**	(8.7 - 17)	**10**	(5.2 - 19)	**8.4**	(4.0 - 22)
PCB-169	94%	**7.3**	(6.5 - 8.1)	**7.3**	(5.2 - 10.4)	**13**	(8.8 - 19)	**11.3**	(6.2 - 28)
PCB-189	92%	**7.8**	(5.5 - 11)	**22**	(16 - 32)	**41**	(24 - 69)	**5.4**	(2.7 - 13)
Reference for dietary intakes		Total diet study samples from 10-16 locations in Japan collected in FY1998-2000[11] (n =45-55)		Total diet study samples from 10-16 locations in Japan collected in FY1998-2000[11] (n =45-55)		Total diet study samples from the Kanto region in Japan collected in FY1998-2000[11] (n =9-12)		Total diet study samples from the Kansai region in Japan collected in FY1977-2000[11] + other information[28-31]	
Reference for concentrations in the human body		Concentrations of congeners in breast milk from 21 locations in Japan collected in FY1998[19] (n =415, women, Age: 25-34)		Concentrations of congeners in blood from 7 locations in Japan collected in FY1998[20] (n =253, men & women, Age: 20-65)		Concentrations of congeners in adipose tissue from the Kanto region of Japan collected in 2000[21] (n =10, women, Age: 40-59)		Concentrations of congeners in breast milk from Osaka, Japan collected in FY1998[19] (n =20, women, Age: 25-34)	
Method for calculations		Half-lives were calculated by Eq. 3 under **steady-state conditions**.		Half-lives were calculated by Eq. 3 under **steady-state conditions**.		Half-lives were calculated by Eq. 3 under **steady-state conditions**.		Half-lives were calculated by a one-compartment kinetic model (Eqs. 4 and 5) under **unsteady-state conditions**.	

* Gastrointestinal absorption (*Abs*) was calculated by the following equation[13]: $1/Abs = 1.01 + 1.55 \times 10^{-9} \times Kow$, where *Kow* is the octanol-water partition coefficient[22-24].

** It was assumed that the distributions of both dietary intakes and concentrations in the human body are lognormal distributions. The representative values of estimated half-lives were calculated by both geometric means of dietary intakes and concentrations in the human body. The values in the parentheses represent 95% confidence intervals for mean values.

*** The values indicate half-lives for women with total body lipid weight of approximately 12000 g. The values in the parentheses were the ranges considering the uncertainties of the concentrations in food and the human body.

References

1 Flesch-Janys D., Becher H., Gurn P., Jung D., Konietzko J., Manz A. and Päpke O. (1996) J. Toxicol. Environ. Health 47, 363-378.
2 Gorski T., Konopka L. and Brodzki M. (1984) Roczn. Pzh. T. 35, 297 (as sited in Van den Berg M., De Jongh J., Poiger H. and Olson J.R. (1994) Critical reviews in toxicology 24, 1-74).
3 Schecter A., Ryan J.J. and Kostyniac P.J. (1990) Chemosphere 20, 911-917.
4 Ryan J.J. and Masuda Y. (1991) Proceedings of 11th international symposium on chlorinated dioxins and related compounds, Dioxin '91: 70.
5 Masuda Y. (1999) Organohalogen Compounds 44, 27-30.
6 Rohde S., Moser G.A., Päpke O. and McLachlan M.S. (1999) Chemosphere 38, 3397-3410.
7 Michalek J.E., Pirkle J.L., Caudill S.P., Tripathi R.C., Patterson D.G. Jr and Needham L.L. (1996) J. Toxicol. Environ. Health 47, 209-220.
8 Needham L.L., Gerthoux P.M., Patterson D.G. Jr, Brambilla P., Pirkle J.L., Tramacere P.I., Turner W.E., Beretta C., Sampson E.J. and Mocoarelli P. (1994) Organolhalogen Compounds 21, 81-85 (as sited in Michalek et al. (1996)[7]).
9 Liem A.K.D. and Theelen R.M.C. (1997) Dioxins. Chemical Analysis, Exposure and Risk Assessment National Institute of Public Health and the Environment, the Netherlands.
10 Van der Molen G.W., Kooijman B.A.L.M., Wittsiepe J., Schrey P., Flesch-Janys D. and Slob W. (2000) J. Expo. Anal. Environ. Epidemiol. 10, 579-585.
11 Ministry of Health and Welfare of Japan (1998, 1999, 2000, 2001) Results of dioxin food contamination survey (in Japanese).
12 Schlummer M., Moser G.A. and McLachlan M.S. (1998) Toxicol. Appl. Pharmacol. 152, 128-137.
13 Moser G.A. and McLachlan M.S. (2001) Chemosphere 45, 201-211.
14 Iida T., Hirakawa H., Matsueda T., Matsueda T., Nakagawa R., Morita K., Hamamura K., Nakayama J., Hori Y., Guo Y.-L.L., Chang F.-M., Hsiao P.-W., Lin K.-C., Yu M.-L., Lai T.-J., Chen S.-J. and Hsu C.-C. (1995) Fukuoka Acta Med. 86, 234-240 (in Japanese).
15 Kitamura K., Nagahashi M., Sunaga M., Watanabe S. and Nagao M. (2001) Journal of Health Science 47, 145-154.
16 Kitamura K., Nagao M., Yamada T., Sunaga M., Hata J. and Watanabe S. (2001) J. Toxic. Sci. 26, 327-336.
17 Iida T., Hirakawa H., Matsueda T., Takenaka S., Yu M.-L. and Guo Y.-L.L. (1999) Chemosphere 38, 981-993.
18 Matsueda T., Hirakawa H., Iida T., Nakamura M. and Nagayama J. (1995) Organohalogen Compounds 26, 219-222.
19 Ministry of Health and Welfare of Japan (1999) Results of survey of dioxins in breast milk in FY1998 (in Japanese).
20 Environment Agency of Japan (1999) Results of survey of dioxins in human tissues, blood, wildlife, and foods in FY1998 (in Japanese).
21 Choi J.W., Miyabara Y., Hashimoto S., Suzuki N. and Morita M. (2002) Chemosphere 47, 591-597.
22 Govers H.A.J. and Krop H.B. (1998) Chemosphere 37, 2139-2152.
23 Hawker D.W. and Connell D.W. (1988) Environ. Sci. Technol. 22, 382-387.
24 Brodsky J. and Ballschmiter K. (1988) Fresenius' Z. Anal. Chem. 331, 295-301.
25 Carrier G., Brunet R.C. and Brodeur J. (1995) Toxicol. Appl. Pharmacol. 131, 267-276.

26 Kreuzer P.E., Csanády Gy.A., Baur C., Kessler W., Päpke O., Greim H. and Filser J.G. (1997) Arch Toxicol 71, 383-400.
27 Ministry of Health and Welfare of Japan (1960-2000) Results of national survey of nutritive conditions (in Japanese).
28 Toyoda M., Matsuda R., Igarashi A. and Saito Y. (1998) Food Sanitation Research 48, 43-65 (in Japanese).
29 PCB special sectional meeting (1972) Environmental Health Report 14, 3-8 (in Japanese).
30 Yao Y., Masunaga S., Takada H. and Nakanishi J. (2002) Environ. Toxicol. Chem. 21, 991-998.
31 Masunaga S., Yao Y., Ogura I., Nakai S, Kanai Y, Yamamuro M. and Nakanishi J. (2001) Environ. Sci. Technol. 35, 1967-1973.