which were apparently lower than those in the present study (Table 3). In general, PCB contamination in general population in Japan is in the moderate to higher levels among developed nations and somewhat higher than that in developing world. Our compilation and data analysis on worldwide human exposure in adipose tissues and liver also demonstrated this feature of contamination (Minh *et al.* 2000a, 2001).

The local and worldwide contamination pattern of OC insecticides in human blood was somewhat different from that of PCBs (Table 4). DDT residue levels in Miyako, Saku and Tottori were comparable to those reported in human blood samples from Sapporo, northern Japan (Takasuga *et al.* 2002). However, the levels were less than those in rural areas in Akita prefecture previously examined by Hanaoka *et al.* (2002). Sasaki *et al.* (1991) also reported elevated concentrations of DDTs in blood of residents from Okinawa in a survey conducted during 1986-88 (Table 4). Mean concentrations of DDTs in Okinawa subjects were 5.5 ng/ml (range 1.5 – 19 ng/ml), which was 4 times greater than that measured in this study for people in Miyako, a district in Okinawa prefecture (Table 4). Although the locations of samples are different between two studies, this can be considered as a hint of significant decline in human exposure to DDTs from Okinawa prefecture. Similar pattern was observed for HCHs and HCB, showing lower levels in the present study than those reported previously in other locations in Japan.

On a global scale, DDT contamination in human blood from Japan was less pronounced when compared to developing countries such as China, India, Egypt, Mexico and Brazil, where heavy usage of DDT for various agricultural and public health purpose have been common during the past four decades. Similar feature of contamination was also observed in adipose tissues and human milk, showing much higher DDT concentrations in populations from developing countries (Minh TB *et al.*, 2000a; Minh NH *et al.* 2004).

Interestingly, CHL concentrations in human from Miyako, Saku and Tottori, Japan were among the highest values reported for the countries examined. Chlordane was banned relatively late (in 1986) in Japan as compared to other persistent OCs and therefore, high contamination of CHLs in Japanese humans still continues to be of considerable concern. Our previous time trend monitoring studies using archived adipose tissues revealed that CHL residues exhibited increasing trend until 1985 (Loganathan *et al.* 1993). Data from Hirai and Tomokuni (1991) and our previous investigation (Minh *et al.* 2001) showed higher concentrations of CHLs in Japanese adipose tissues examined in 1999 than those collected in 1989. The steady trend of CHL contamination in Japanese human samples during the last four decades reflect the remarkable increase in the usage of CHLs as termite control agent before it has been officially banned in 1986. Statistical data from the Ministry of the Environment, Japan showed a significant increase in the amount of CHL imported to Japan, and the import reached the maximum amount of approximately > 2000 tons in 1985 (Ministry of the Environment, Japan 2002). Therefore, although CHL was banned in Japan in 1986, elevated contamination of these compounds still remain in Japanese human samples.

Major route of human exposure to persistent OC compounds is via dietary intake (mainly fatty food, fish, meat and dairy product). Therefore, it is worthy to compare the residue concentrations of OCs in human tissues in relation to the dietary intake. A compilation of OC concentrations in human blood samples and dietary intake via foodstuffs of different countries is given in Figure 2. Data for dietary intake were cited from an extensive review article done by Kannan *et al.* (1997). Since dietary intakes can be substantially different among locations and time of investigations as a result of effective restriction and prohibition of OCs usage, data on blood concentrations and intakes reported at similar periods of time were cited for comparison as far as possible. In general, intakes of PCBs, DDTs and HCHs more or less reflect the contamination status in human blood samples (Figure 2). Some inconsistent results observed in intake and blood levels of OCs in Finland and United States (for PCBs, HCHs and DDTs), which could be due to the different foods and seafood consumption, or kinds of food used for estimating the dietary intakes. It can be emphasized that CHL compounds have lower dietary intakes via foodstuffs. CHL compounds were used in the United States and Japan as termite control agent, and technical chlordane was sprayed directly to the households to eradicate termite. In the United States, it was estimated that over 80 million people lived in homes treated with CHLs and these compounds were detected in the indoor air of homes even 15 years after treatment (ATSDR 1994). In Japan, a previous occupational investigation revealed immediate increase in CHL residues in blood and sebum of residents of the treated houses and pest control operators (Wariishi and Nishimura 1989). Direct exposure via indoor air and house walls (inhalation or skin contact) is a major route of intake of CHLs into human body, in addition to the dietary intake via foods. These facts explain the differences in CHL residue concentrations in human blood and dietary intake estimated via foodstuffs as described in Figure 2.

Accumulation pattern and time trend of chlordane compounds in Japanese human blood samples

In this study, *p,p'*-DDE and β-HCH were predominant chemicals among DDT compounds and HCH isomers, respectively. In all the plasma samples analyzed, they account for > 90 % of the total DDT and HCH concentrations. This result is common in human samples and is similar to the pattern observed in adipose tissues from Tokyo, Japan, which we previously examined (Minh *et al.* 2001). Therefore, in this section, we examined the accumulation pattern of chlordane compounds in comparison with data from previous investigations to understand the time trend of contamination in Japanese human blood. Sasaki *et al.* (1991) in a 1986-88 survey reported CHL residues in human blood from Okinawa prefecture; and for this study, data are available for only three compounds, oxychlordane, *trans*- and *cis*-nonachlor. Wariishi *et al.* (1986) reported CHL residues in human blood from Tokushima, a small city located at the southern part of Japan (Shikoku island), and compared CHL exposure in subjects having no special exposure to CHLs, and those who were exposed to CHLs following termite control treatment in their houses. Subsequent occupational study done by the same authors (Warrishi and Nishiyama 1989) provided an extensive investigation on the changes in CHL blood levels in pest control operators from Tokushima, who were occupationally exposed to elevated CHL concentrations. Residents from Okinawa, who were not occupationally exposed, are considered as controls in this investigation. Total residue levels of CHLs and proportions of the related compounds (oxychlordane, *trans*-chlordane, *cis*-chlordane, *trans*-nonachlor and *cis*-nonachlor) to total CHL concentrations reported in these investigations and in the present study were compared to understand the geographical differences in accumulation pattern, the possible implications of occupational exposure on the changes of accumulation pattern, and the time trend of CHLs in Okinawa residents during the last decade (Figure 3). Since lipid-normalized concentrations were not reported in the previous studies (Wariishi *et al.* 1986; Wariishi and Nishiyama 1989; Sasaki *et al.* 1991), concentrations on a wet weight basis were used for comparison. In general, oxychlordane and *trans*-nonachlor were predominant compounds. In Miyako, Okinawa, proportions of these chemicals were higher than those in Saku and Tottori (Figure 3). Similar pattern was noticed in Tokushima in an investigation during the period of 5 years after the CHL treatment for termite control (Figure 3) (Wariishi *et al.* 1986). Occupational analysis on subjects from Tokushima revealed substantial differences in residues concentrations and pattern (proportions to total CHL concentrations) in pest control operators (workers who are occupationally exposed to elevated CHL levels) and in those not occupationally exposed (controls). Proportions of oxychlordane in workers were much lower than those in controls (Wariishi and Nishiyama 1989). Oxychlodane is the terminal metabolic product of other chlordane compounds such as *trans*-chlordane and *cis*-chlordane (Wariishi *et al.* 1986). Immediate changes in the levels of *trans*-nonachlor and *trans*-chlordane in human blood were observed after termite control treatment and *trans*-nonachlor residues remained relatively constant even after 5 months from occupational exposure (Wariishi and Nishiyama

1989). This phenomenon can explain the higher proportions of *trans*-nonachlor and oxychlordane in high exposure groups.

Data on human plasma in Miyako analyzed in this study were compared to those in residents from Okinawa reported by Wariishi and Nishiyama (1989) and Sasaki *et al.* (1991) to discern the time trend of CHLs during the last decade (Figure 3). Residue concentrations of total CHLs declined substantially; about 2 times lower than in 1986-88 survey (Sasaki *et al.* 1991) and 10 times lower than in 1980-85 investigation (Warrishi and Nishiyama 1989). Although the backgrounds of samples and analytical methods of each study are different, results of this paper suggested that the CHL residue concentrations in human blood from Okinawa, Japan exhibited a declining trend during the last decade. Proportions of *trans*-chlordane and *cis*-chlordane, the two major components of technical chlordane, in 1999 survey (present study) were also apparently lower than those in the previous investigation (Wariishi and Nishiyama 1989). Loganathan *et al.* (1993) reported a relatively rapid increasing trend in CHL concentrations in human adipose tissues from Japan during 1955-1985, which is parallel with the remarkable increase in the amount of CHLs imported for agricultural usage and termite control. The amount of technical CHL imported to Japan for agricultural usage increased about 3 times during 1963-68. Subsequently, from 1979 a remarkable increase in import amount of CHL for termite control was noticed, reached a maximum level of > 2,000 tons in 1985 before it was officially banned in 1986 (Ministry of the Environment, Japan 2002). Thus, monitoring studies using human samples as bio-indicators clearly reflect the status of CHL usage and the effect of the ban of CHLs on the accumulation pattern in human blood.

PCB isomer pattern

Isomer-specific analysis of PCB revealed the presence of 60 congeners. CB-118, 138, 153 and 180 were predominant congeners, and this result is common for human samples. In females, we found that relative concentrations of higher chlorinated congeners such as hepta- and octachlorobiphenyls were lower than those in males (although no statistical differences were noticed), suggesting a possibility of preferential elimination of higher chlorinated biphenyls in females (Figure 4). In contrast, proportions of lower chlorinated biphenyl congeners (tetra- and pentachlorobiphenyls) were higher in females than those in males. This male – female difference in PCB patterns was also observed in Dall's porpoises collected from off Pacific coasts of Japan (Subramanian *et al.* 1988), suggesting the similarity in accumulation kinetics of PCBs in higher trophic animals.

Age and sex dependent accumulation

To understand the accumulation kinetics of persistent OC compounds in healthy persons and diseased persons, we compared the age and gender differences in OC residue levels in the subjects examined in the present study and in autopsied adipose tissues of dead subjects who lived in Tokyo and nearby areas, which were analyzed in our previous study employing the same analytical methods (Minh *et al.* 2001) (Table 5 and 6, Figure 5 and 6). Although human tissues were different among two studies, it has been reported previously that blood residues indeed reflect adipose tissue concentrations, because significant correlations of OC concentrations in blood and adipose tissues were observed (review by Pelletier *et al.* 2003). In general, concentrations of OC compounds tended to increase with age in both males and females, which are similar to those found in human adipose tissue previously reported (Minh *et al.* 2001) (Figure 5 and 6). This result indicates long term accumulation of persistent and highly bio-accumulative OC compounds in humans despite phasing out of the usage of these chemicals in Japan more than 30 years ago. Similar age-dependent accumulation was also observed in human samples from other countries such as Netherlands (Greve and van Zoonen 1990), Spain (Ferrer *et al.* 1992), Italy (Focardi *et al.* 1986), Belgium (Voorspoels *et al.*, 2002) and Korea (Kang *et al.* 1997).

A Spearman rank correlation analysis revealed that PCB, DDT and TCPMe concentrations in blood from Miyako and in adipose tissues from Tokyo correlated significantly with age (Table 6). Interestingly, TCPMe showed the highest correlation with age in all the locations examined (Table 6), which further indicate high bio-accumulation potential of this compound in humans. This is quite consistent to the result observed for adipose tissue (Minh *et al.* 2001). Good positive correlation between concentrations and age suggest that body growth seems to be an important factor influencing the TCPMe burdens in humans. In marine mammals, it has been also suggested that differences in metabolic capacity among different species play a crucial role in the accumulation kinetics of TCPMe (Minh *et al.* 2000b).

Apparent gender differences were observed in samples from three locations, with higher concentrations in males than in female. A Mann-Whitney *U* test showed significant gender differences for PCBs in Miyako and Saku, for CHLs and TCPMe in Saku and Tottori (Table 5, Figure 5 and 6). In the previous study on sex-dependent accumulation in autopsied adipose tissues of patients, no apparent gender differences were noticed (Minh *et al.* 2001). Based on these different results, we suggest that accumulation kinetics of persistent OC compounds may be different among healthy and diseased persons. In diseased subjects, nutrient deficient conditions were observed with extremely low lipid content in adipose tissue, enhancing concentrations of OCs in the remaining lipids (Minh *et al.* 2001). Mobilization of stored contaminants from adipose tissue takes place during starvation or lactation and as a result of diseases (Thomas and Colborn 1992). It has been reported that increases in plasma OC concentrations significantly correlated with decreased in fat mass, suggesting that OCs are released into the blood during lipid mobilization (Pelletier *et al.* 2003). Bioaccumulation kinetics (age- and sex-dependent accumulation) of persistent OCs in human body is therefore strongly influenced by the lipid deficiency and weight loss, which is commonly observed in diseased persons.

Conclusions and Recommendations

To our knowledge, this is the first report on the specific accumulation of persistent OCs including TCPMe in human blood from Japan. Our result demonstrated elevated accumulation of PCBs and CHLs in Japanese humans, despite the ban on these chemicals for more than two decades in Japan. Given that limited data on OC residues in Japanese human blood, the result of this study can be a considerable contribution to the database of human exposure to persistent OCs in Japan. On the basis of the results of this study, we suggest that a systematic national survey on persistent organic pollutants (particularly PCBs, DDTs and CHLs) should be conducted by the Ministry of Health, Labor and Welfare, Japan.

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Table 1. Concentrations (ng/g lipid wt) of persistent organochlorines in human blood from Miyako, Saku and Tottori, Japan.

Sample Code	Sex	Age	PCBs	DDTs	HCHs	CHLs	HCB	TCPMs
Miyako (n = 23)								
210	M	27	160	100	16	27	6.0	< 0.1
243	M	28	180	88	13	28	4.6	< 0.1
203	M	30	230	85	16	55	8.5	2.0
247	M	38	440	190	33	60	8.2	2.6
230	M	41	620	1400	95	150	20	8.1
250	M	43	240	56	20	70	7.5	5.7
204	M	46	240	620	72	70	8.3	< 0.1
206	M	46	430	500	85	150	12	< 0.1
219	M	49	250	110	7.6	24	4.8	4.7
225	M	51	340	230	39	59	8.7	4.7
205	M	55	380	990	20	92	6.6	< 0.1
229	M	57	340	350	120	77	5.8	5.6
221	M	58	360	280	24	43	5.2	5.9
216	F	24	200	230	23	25	7.1	< 0.1
208	F	25	120	76	22	150	8.9	0.7
246	F	25	180	80	17	34	6.5	1.6
245	F	30	170	120	40	53	8.1	2.1
226	F	31	160	73	36	58	7.8	1.8
234	F	35	320	150	65	74	11	5.9
254	F	38	290	240	28	29	11	5.1
244	F	42	290	400	24	77	7.2	5.4
222	F	49	170	350	32	99	6.1	3.5
217	F	52	360	300	130	110	12	6.7
Mean[a]		40	280	310	43	70	8.3	3.2
Median		41	250	230	28	60	7.8	3.1
Geometric mean		38	260	210	32	60	7.9	1.4
Range		24 - 58	120 - 620	56 - 1400	7.6 - 130	24 - 150	4.6 - 20	< 0.1 - 8.1
Saku (n = 32)								
335	M	30	240	64	35	13	5.1	0.82
301	M	34	580	130	15	22	15	1.8
329	M	39	390	79	1.7	23	6.2	0.72
332	M	40	350	300	27	31	14	2.5
345	M	40	350	390	22	65	7.9	2.3
362	M	46	720	360	37	47	4.6	3.4
316	M	51	730	900	68	68	32	3.9
323	M	51	780	180	0.81	29	6.7	2.3
328	M	52	440	270	23	36	11	2.1
343	M	52	590	440	32	34	18	3.7
339	M	53	480	460	37	55	24	3.7
360	M	55	530	150	100	44	20	2.4
348	M	57	210	35	32	16	3.3	2.1
327	M	59	450	370	41	25	9.1	2.6
308	M	59	990	2700	64	79	27	6.2
306	F	28	260	230	44	17	14	0.62
356	F	32	160	140	17	4.8	4.1	< 0.1
349	F	35	440	840	49	26	12	1.2
359	F	37	220	75	28	20	8.3	1.1
312	F	43	270	210	54	21	14	1.2
366	F	48	560	1300	53	42	13	2.0
338	F	51	270	230	35	19	14	1.4
310	F	52	310	130	36	31	15	0.54
321	F	53	250	320	35	23	15	3.4
342	F	53	290	130	22	19	17	1.9
354	F	54	240	180	33	21	13	0.64
358	F	54	140	310	22	21	11	0.56
309	F	54	260	290	24	26	19	1.9
367	F	56	320	64	22	47	3.3	1.5
313	F	57	310	200	39	34	5.8	1.3
303	F	58	450	190	64	35	8.2	1.5
317	F	59	320	32	30	26	< 1.0	4.5
Mean		48	400	370	36	32	12	2.1
Median		52	340	220	34	26	13	1.9
Geometric mean		47	360	220	28	28	10	1.6
Range		28 - 59	140 - 990	32 - 2700	0.81 - 100	4.8 - 79	< 1.0 - 32	< 0.1 - 6.2
Tottori (n = 22)								
470	M	30	200	34	9.8	24	5.1	0.42
416	M	37	250	96	24	32	9.5	1.2
420	M	45	650	210	37	49	16	3.8
407	M	47	560	250	95	46	12	1.8
431	M	48	350	200	26	33	11	2.1
405	M	52	420	85	26	38	14	2.6
408	M	56	300	200	38	34	10	2.9
469	M	56	520	420	42	68	8.9	3.4
450	M	57	550	190	69	24	11	4.9
429	M	59	780	440	46	67	13	4.2
471	F	30	220	50	17	12	5.9	1.4
454	F	35	480	380	64	30	12	1.8
418	F	39	230	130	41	21	11	2.2
440	F	41	240	89	9.2	14	7.6	0.9
446	F	43	270	70	34	44	6.4	4.8
426	F	45	490	490	32	28	10	2.8
438	F	48	200	140	24	16	8.2	2.1
457	F	49	290	210	32	27	9.5	1.9
451	F	49	400	75	34	17	9.1	1.7
449	F	50	510	350	44	28	13	2.0
458	F	55	400	230	42	19	9.5	0.9
436	F	59	210	190	28	18	8.1	2.7
Mean		47	390	210	37	31	10	2.4
Median		48	380	200	34	28	9.8	2.1
Geometric mean		46	360	160	32	28	9.7	2.1
Range		30 - 59	200 - 780	34 - 490	9.2 - 95	12 - 68	5.1 - 16	0.42 - 4.9

[a]Concentration values below the detection limit were assigned as equal to detection limit for calculation of mean, median and geometric mean.

Table 2. Production and usage of persistent organochlorine compounds in Japan

Organochlorine compound	Approximate amount of production/usage (tons)	Year of starting usage/production	Year of ban	Source of information/Reference
PCBs	58,787/54,001	early 1950s	1972	Ministry of the Environment, Japan (2002)
DDTs	45,000	1945	1971	Ministry of the Environment, Japan (2002)
HCHs	400,000	1950	1972	Li (1999)
CHLs	> 2,000	1955	1986	Ministry of the Environment, Japan (2002)
HCB	4,000 (peak annual demand)	NA[a]	1979	Ministry of the Environment, Japan (2002)

[a]NA: no data available.

Table 3. Comparison of the concentrations of polychlorinated biphenyl congeners (PCBs) in human blood from different locations in the world.[a]

Location	Year	Sex	Age (year)	*n*	Tissue	Unit	CB-118	CB-138	CB-153	CB-180	Total PCBs	Reference	Remark
Miyako, Okinawa, Japan	1999	M&F[b]	24 - 58	23	plasma	ng/g lipid	12 (4.3 - 39)[c]	33 (21 - 140)	50 (21 - 140)	43 (13 - 99)	280 (120 - 620)	Present study	Total PCBs: sum of 60 congeners
						ng/ml	0.048 (0.017 - 0.16)	0.13 (0.084 - 0.56)	0.2 (0.084 - 0.56)	0.17 (0.052 - 0.40)	1.1 (0.48 - 2.5)		
Saku, Nagano, Japan	1999	M&F	28 - 59	32	plasma	ng/g lipid	23 (4.3 - 65)	53 (11 - 150)	77 (17 - 210)	55 (11 - 150)	400 (140 - 990)	Present study	Total PCBs: sum of 60 congeners
						ng/ml	0.09 (0.017 - 0.25)	0.2 (0.043 - 0.59)	0.3 (0.067 - 0.84)	0.21 (0.043 - 0.59)	1.6 (0.55 - 3.9)		
Tottori, Tottori, Japan	1999	M&F	30 - 59	22	plasma	ng/g lipid	20 (9.1 - 50)	50 (20 - 100)	80 (37 - 180)	66 (33 - 150)	390 (200 - 780)	Present study	Total PCBs: sum of 60 congeners
						ng/ml	0.08 (0.036 - 0.2)	0.2 (0.08 - 0.4)	0.32 (0.15 - 0.72)	0.26 (0.13 - 0.6)	1.6 (0.8 - 3.1)		
Sapporo, Japan	NA	M&F	37-48	18	whole blood	ng/g lipid	NA[d]	NA	NA	NA	270 (62 - 970)	Takasuga et al. (2002)	NA
Anqing, China	1998	F	25 ± 1.2[e]	15	serum	ng/ml	NA	NA	NA	NA	0.2 ± 0.1	Korrick et al. (2001)	Total PCBs: sum of 67 congeners
Great Lakes Region, US	1993	M&F	36 - 76	30	serum	ng/ml	0.4 (0.02 - 1.1)	0.8 (0.1 - 2.2)	1.1 (0.3 - 2.9)	0.4 (0.09 - 1.0)	5.2 (1.2 - 15)	Anderson et al. (1998)	Total PCBs: sum of 30 congeners
New York, US	1991	F	45 - 85	192	serum	ng/ml	NA	NA	NA	NA	4.1 ± 2.2	Moysich et al. (2002)	Total PCBs: sum of 69 congeners
Long Island, NY, US	1996-97	F	> 20	1556	serum	ng/g lipid	56 ± 2.0	85 ± 2.5	150 ± 1.7	79 ± 1.8	370 ±1.7	Gammon et al. (2002)	Total PCBs: sum of 4 congeners
US, 11 states	1989-90	F	30-55	370	plasma	ng/g lipid	68	97	110	75	540	Laden et al. (2001)	Total PCBs: sum of 16 congeners
North Carolina, US	1993-1995	F	28-74	68	plasma	ng/ml	0.34	0.44	0.49	0.34	1.4	DeVoto et al. (1997)	Total PCBs: sum of 26 congeners
Wisconsin, US	1991	M&F	20-71	100	serum	ng/ml	0.62	1.4	1.7	1	6.9	DeVoto et al. (1997)	Total PCBs: sum of 26 congeners
Canadian Arctic	1994-99	F	15 - 45	385	plasma	ng/g lipid	8.6 (<2.2 - 71)	27 (<2.2 - 350)	48 (2.2 - 890)	23 (2.2 - 450)	140 (12 - 2300)	Walker et al. (2003)	Total PCBs: sum of 14 congeners
Greenland Arctic	1997	M	19-60	15	plasma	ng/ml	1.0 (0.18 - 4.3)	6.0 (1.0 - 19)	14 (2.4 - 44)	9.6 (1.4 - 31)	41 (6.6 - 130)	Van Oostdam & Tremblay (2003)	Total PCBs: sum of 14 congeners
Alaskan Arctic	2001	M&F	18-91	166	serum	ng/ml	NA	NA	NA	NA	7.7 (nd[f] - 54)	Van Oostdam & Tremblay (2003)	Total PCBs: sum of 36 congeners
Russian Arctic	1999	F	NA	30	plasma	ng/ml	0.29 (0.1 - 1.0)	0.42 (0.12 - 1.5)	0.39 (0.11 - 1.7)	0.13 (0.04 - 1.0)	1.7 (0.6 - 6.8)	Van Oostdam & Tremblay (2003)	Total PCBs: sum of 13 congeners
Norwegian Arctic	1999	F	NA	50	plasma	ng/ml	0.11 (0.01 - 0.67)	0.79 (0.24 - 2.3)	0.47 (0.17 - 1.2)	0.4 (0.13 - 0.84)	2.3 (0.8 - 6.2)	Van Oostdam & Tremblay (2003)	Total PCBs: sum of 11 congeners
Finland	1996-98	F	NA	13	plasma	ng/ml	0.07 (0.04 - 0.11)	0.3 (0.15 - 0.4)	0.43 (0.21 - 0.61)	0.24 (0.15 - 0.33)	1.5 (0.8 - 2.0)	Van Oostdam & Tremblay (2003)	Total PCBs: sum of 14 congeners
Sweden, central region	NA	M	40 - 74	120	serum	ng/g lipid	42 (4.3 - 140)	140 (3.1 - 340)	300 (23 - 630)	220 (71 - 480)	750 (150 - 1500)	Glynn et al. (2000)	Total PCBs: sum of 10 congeners
Sweden	1996-97	F	50 - 74	205	serum	ng/g lipid	43 (5.0 - 180)	100 (18 - 270)	220 (60 - 610)	150 (56 - 400)	550 (150 - 1600)	Glynn et al. (2003)	Total PCBs: sum of 7 congeners
Netherlands	1990-92	F	NA	418	plasma	ng/ml	0.16	0.60	0.91	0.54	2.2	Koopman-Esseboom et al. (1994)	Total PCBs: sum of 4congeners
Flanders, Belgium	1996-98	F	24-42	96	serum	ng/g lipid	27 ± 16	71 ± 33	91 ± 39	70 ± 31	260	Pauwels et al. (2000)	Total PCBs: sum of 4 congeners
Belgium	2001	M&F	20-54	132	serum	ng/g lipid	13	64	99	60	240	Voorspoels et al. (2002)	Total PCBs: sum of 4 congeners
Wurzburg, Germany	1992-95	M&F	NA	309	whole blood	ng/ml	NA	0.39 ± 0.22	0.49 ± 0.29	0.24 ± 0.16	1.2	Loffler et al. (2000)	Total PCBs: sum of 4 congeners

[a]Cited values were rounded to 2 significant digits for comparison. [b]M, Male, F, Female. [c]Range concentration. [d]NA, no data available. [e]Mean ± standard deviation (SD). [f]nd, not detected; detection limit was not reported.

Table 4. Comparison of organochlorine insecticide residue levels in human blood from various locations in the world.[a]

Location	Year	Sex	Age (year)	n	Tissue	Unit	DDTs	HCHs	CHLs	HCB	Ref.[b]	Remark
Miyako, Okinawa, Japan	1999	M&F[c]	24 - 58	23	plasma	ng/g lipid	310 (56 - 1400)[d]	43 (7.6 - 130)	70 (24 - 150)	8.3 (4.6 - 12)	(1)	DDTs: p,p'-DDE; p,p'-DDD and p,p'-DDT; HCHs: α-, β- and γ-HCH
						ng/ml	1.3 (0.22 - 5.6)	0.17 (0.03 - 5.2)	0.28 (0.096 - 0.6)	0.033 (0.018 - 0.048)		CHLs: trans- and cis-nonachlor, trans- and cis-chlordane, and oxychlordane
Saku, Nagano, Japan	1999	M&F	28 - 59	32	plasma	ng/g lipid	370 (32 - 2700)	36 (0.81 - 100)	32 (4.8 - 79)	12 (<1.0 - 32)	(1)	DDTs: p,p'-DDE; p,p'-DDD and p,p'-DDT; HCHs: α-, β- and γ-HCH
						ng/ml	1.4 (0.12 - 11)	0.14 (0.0032 - 0.39)	0.12 (0.019 - 0.31)	0.047 (<0.004 - 0.12)		CHLs: trans- and cis-nonachlor, trans- and cis-chlordane, and oxychlordane
Tottori, Tottori, Japan	1999	M&F	30 - 59	22	plasma	ng/g lipid	210 (34 - 490)	37(9.2 - 95)	3.0 (12 - 68)	10 (5.1 - 16)	(1)	DDTs: p,p'-DDE; p,p'-DDD and p,p'-DDT; HCHs: α-, β- and γ-HCH
						ng/ml	0.84 (0.14 - 2.0)	0.15 (0.037 - 0.38)	0.12 (0.05 - 0.27)	0.04 (0.02 - 0.064)		CHLs: trans- and cis-nonachlor, trans- and cis-chlordane, and oxychlordane
Sapporo, Japan	NA	M&F	37-48	18	whole blood	ng/g lipid	350 (61 - 1600)	140 (14 - 710)	43 (7.6 - 150)	18 (2.7 - 120)	(2)	NA
Akita, Japan	1999	M&F	41 - 69	41	serum	ng/g lipid	3000 (430 - 15000)	260 (24 - 710)	NA[e]	110 (9.5 - 330)	(3)	DDTs: p,p'-DDE, and p,p'-DDT; HCHs: β-HCH only
						ng/ml	6.3 (0.9 - 31)	0.54 (0.05 - 1.5)	NA	0.24 (0.02 - 0.7)		DDTs: p,p'-DDE, p,p'-DDD and p,p'-DDT; HCHs: β-HCH only
Akita, Japan	1986-88	M&F	54 - 78	12	whole blood	ng/ml	4.3 (1.9-8.2)	2.2 (0.8 - 5.5)	0.30 (0.15 - 0.96)	NA	(4)	DDTs: p,p'-DDE only; HCHs; β-HCH only;
Okinawa, Japan	1986-88	M&F	19 - 67	11	whole blood	ng/ml	5.5 (1.5 - 19)	1.8 (0.7 - 4.2)	0.88 (0.37 - 2.8)	NA	(4)	CHLs: trans- and cis-nonachlor, heptachlor and oxychlordane
Anqing, China	1998	F	25 ± 1.2[f]	15	serum	ng/ml	13 ± 6	NA	0.01 ± 0.01	0.3 ± 0.1	(5)	DDTs: p,p'-DDE, p,p'-DDD, p,p'-DDT, o,p'-DDE and o,p'-DDT; CHLs: oxychlordane only
Jaipur city, India	1997-98	F	11 - 50	100	whole blood	ng/ml	6900 ± 550	3900 ± 350	1400 ± 150	NA	(6)	DDTs: p,p'-DDE, p,p'-DDD and p,p'-DDT; HCHs: α-, β- and γ-HCH; CHLs: heptachlor only
Nainital, India	2000	M&F		39	whole blood	ng/ml	4600 (1000-13000)	1300 (250 - 7500)	NA	NA	(7)	DDTs: p,p'-DDE, p,p'-DDD, p,p'-DDTand o,p'-DDT; HCHs: α-, β- and γ-HCH
Egypt, rural area		F	18 - 50	76	serum	ng/ml	18 (0.3 - 140)	2.6 (0.3-18)	NA	NA	(8)	DDTs: p,p'-DDE, p,p'-DDD and p,p'-DDT; HCHs: β-HCH only
Egypt, urban area		F	18 - 50	46	serum	ng/ml	9.9 (0.7-62)	1.3 (0.3-9.5)	NA	NA	(8)	DDTs: p,p'-DDE, p,p'-DDD and p,p'-DDT; HCHs: β-HCH only
Veracruz, Mexico	1997-98	F	18 - 44	60	serum	ng/ml	16 ± 31	1.6 ± 2.0	NA	1.0 ± 0.8	(9)	DDTs: p,p'-DDE, p,p'-DDD, p,p'-DDT, and o,p'-DDT; HCHs: α-, β- and γ-HCH
Rio de Janeiro, Brazil	1997	F	16 - 49	64	plasma	ng/ml	3.0 (0.52 - 18)	1.0 (0.22 - 5.1)	0.18 (0.015 - 0.75)	0.084 (0.008 - 0585)	(10)	DDTs: p,p'-DDE, p,p'-DDD and p,p'-DDT; HCHs: α-, β- and γ-HCH; CHLs: heptachlor only
Great Lakes Region, US	1993	M&F	36 - 76	30	serum	ng/ml	5.6 (0.68 - 25)	0.05 (0.04 - 1.2)	1.1 (0.16-3.6)	0.1 (0.02-0.2)	(11)	DDTs: p,p'-DDE; p,p'-DDT and o,p'-DDT; HCHs: β-HCH only; CHLs: oxychlordane, heptachlor epoxide, trans- and cis-nonachlor
New York, US	1991	F	45 - 85	192	serum	ng/ml	11 ± 10	NA	NA	0.42 ± 0.19	(12)	DDTs: p,p'-DDE only
Canadian Arctic	1994-99	F	15 - 45	385	plasma	ng/g lipid	200 (13 - 4100)	25 (< 4.3 - 4200)	66 (8.6 - 1200)	38 (2.2 - 490)	(13)	DDTs: p,p'-DDE and p,p'-DDT; HCHs: β-HCH only; CHLs: α-, γ-chlordane, trans-, cis-nonachlor, heptachlor epoxide, and oxychlordane
Greenland Arctic	1997	M	19-60	15	plasma	ng/ml	11 (1.6 - 33)	0.56 (0.17 - 1.8)	6.9 (0.81 - 65)	1.5 (0.4 - 3.9)	(14)	DDTs: p,p'-DDE and p,p'-DDT; HCHs: β-HCH only; CHLs: oxychlordane and trans-nonachlor
Alaskan Arctic	2001	M&F	18-91	166	serum	ng/ml	14 (nd[g] - 160)	0.35 (nd - 2.6)	2.4 (nd - 18)	NA	(14)	DDTs: p,p'-DDE and p,p'-DDT, HCHs: β-HCH only; CHLs: oxychlordane, trans-nonachlor and heptachlor epoxide
Russian Arctic	1995-96	F	NA	18	plasma	ng/ml	1.2 (0.43 - 2.1)	0.35 (0.02 - 0.76)	0.09 (0.04 - 0.15)	0.2 (0.02 - 0.71)	(14)	DDTs: p,p'-DDE and p,p'-DDT; HCHs: β-HCH only; CHLs: trans-nonachlor only
Norwegian Arctic	1999	F	NA	50	plasma	ng/ml	0.95 (0.20 - 5.1)	0.05 (0.02 - 0.36)	0.14 (0.06 - 0.43)	NA	(14)	DDTs: p,p'-DDE only, HCHs: β-HCH only; CHLs: oxychlordane and trans-nonachlor
Finland	1996-98	F	NA	13	plasma	ng/ml	0.6 (0.21 - 0.83)	0.07 (0.02 - 0.09)	0.08 (0.04 - 0.12)	0.19 (0.12 - 0.31)	(14)	DDTs: p,p'-DDE and p,p'-DDT; HCHs: β-HCH only; CHLs: oxychlordane and trans-nonachlor
Sweden	1996-97	F	50 - 74	205	serum	ng/g lipid	490 (32 - 2500)	51 (7 - 744)	36 (9 - 120)	65 (15-350)	(15)	DDTs: p,p'-DDE only; HCHs: β-HCH only; CHLs: oxychlordane and trans-nonachlor
Sweden, central region	NA	M	40 - 74	120	serum	ng/g lipid	840 (25 - 4100)	52 (12 - 210)	44 (12 - 130)	83 (23 - 1500)	(15)	DDTs: p,p'-DDE, p,p'-DDD, p,p'-DDT, o,p'-DDE and o,p'-DDT; HCHs: α-, β- and γ-HCH; CHLs: trans-nonachlor and oxychloridane
Wurzburg, Germany	1992-95	M&F	NA	309	whole blood	ng/ml	1.5 ± 1.3	0.27 ± 0.16	NA	1.5 ±1.5	(16)	DDTs: p,p'-DDE, p,p'-DDD, p,p'-DDT, o,p'-DDE, o,p'-DDD and o,p'-DDT; HCHs: β- and γ-HCH
Flanders, Belgium	1996-98	F	24 - 42	101	serum	ng/g lipid	1300 ± 1500	NA	NA	NA	(17)	DDTs: p,p'-DDE only

[a]Cited values were rounded to 2 significant digits for comparison. [b]Reference: (1) present study; (2) Takasuga et al. (2002); (3) Hanaoka et al. (2002); (4) Sasaki et al. (1991); (5) Korrick et al. (2001); (6) Rathore et al. (2002); (7) Dua et al. (2002); (8) Soliman et al. (2003); (9) Waliszewski et al. (2001); (10) Sarcinelli et al. (2003); (11) Anderson et al. (1998); (12) Moysich et al. (2002); (13) Walker et al. (2003); (14) Van Oostdam and Tremblay (2003); (15) Glynn et al. (2003); (16)Loffler et al. (2000); (17)Pauwels et al. (2000).

[c]M,male, F,female. [d]Range concentration. [e]NA, no data available. [f]Mean ± standard deviation (SD). [g]nd, not detected; detection limit was not reported.

Table 5. Gender differences in organochlorine accumulation in human blood from Miyako, Saku and Tottori and in human adipose tissues from Tokyo and nearby areas, Japan[a].

Location	Age	*n*	Concentration (ng/g lipid wt) [median, mean ± SD (range)]b					
			PCBs	DDTs	HCHs	CHLs	HCB	TCPMe
Miyako								
Male	44 ± 11 (27 - 58)	13	340, 320 ± 130 (160 - 620)*	230, 380 ± 410 (56 - 1400)	24, 43 ± 37 (7.6 - 120)	60, 70 ± 41 (24 - 150)	7.5, 8.2 ± 4.1 (4.6 - 20)	2.6, 3.1 ± 2.8 (<0.1 - 8.1)
Female	35 ± 10 (24 - 52)	10	190, 230 ± 81 (120 - 360)*	190, 200 ± 120 (73 - 400)	30, 42 ± 34 (17 - 130)	66, 71 ± 40 (25 - 150)	8.0, 8.6 ± 2.1 (6.1 - 12)	2.8, 3.3 ± 2.3 (<0.1 - 6.7)
Saku								
Male	48 ± 9.2 (30 - 59)	15	480, 520 ± 210 (210 - 990)**	300, 460 ± 660 (35 - 2700)	32, 36 ± 26 (0.81 - 100)	34, 39 ± 20 (13 - 79)*	11, 14 ± 8.9 (3.3 - 32)	2.4, 2.7 ± 1.4 (0.72 - 6.2)**
Female	48 ± 9.7 (25 - 59)	17	270, 300 ± 100 (140 - 560)**	200, 290 ± 320 (32 - 1300)	35, 36 ± 13 (17 - 64)	23, 25 ± 10 (4.8 - 47)*	13, 11 ± 5.1 (1.0 - 19)	1.3, 1.5 ± 1.1 (<0.1 - 4.5)**
Tottori								
Male	49 ± 9.4 (30-59)	10	470, 460 ± 190 (200 - 780)	200, 210 ± 130 (34 - 440)	38, 41 ± 25 (9.8 - 95)	36, 42 ± 16 (24 - 68)**	11, 11 ± 3.0 (5.1 - 16)	2.9, 2.7 ± 1.4 (0.42 - 4.9)
Female	45 ± 8.2 (30 - 59)	12	280, 330 ± 120 (200 - 510)	170, 200 ± 140 (50 - 490)	33, 33 ± 14 (9.2 - 64)	20, 23 ± 8.9 (12 - 44)**	9.3, 9.2 ± 2.0 (5.9 - 13)	2.0, 2.1 ± 1.0 (0.9 - 4.8)
Tokyo and nearby areas[c]								
Male	57 ± 19 (17 - 81)	12	1700, 2000 ± 1600 (230 - 6600)	1800, 1800 ± 1200 (200 - 4800)	230, 340 ± 270 (47 - 900)*	250, 410 ± 550 (90 - 2100)	36, 44 ± 25 (21 - 110)*	13, 17 ± 11 (2.7-44)
Female	70 ± 14 (48 - 87)	8	2400, 2200 ± 880 (400 - 3200)	1800, 2900 ± 2800 (160 - 8100)	850, 1200 ± 1000 (130 - 3200)*	340, 400 ± 240 (42 - 730)	86, 85 ± 41 (17 - 160)*	18, 21 ± 11 (5.7-39)

[a]Mann Whitney's *U* Test was used for evaluating gender differences in OC concentrations: *$p < 0.05$, **$p < 0.005$. [b]Median, mean ± standard deviation (SD), range in parentheses.

[c]Data for adipose tissues in diseased persons from Tokyo and nearby areas were cited from our previous study (Minh et al. 2001)