Estimation of dioxin risk to Japanese from the past to the future

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Abstract

Transport processes of seventeen 2,3,7,8-chlorinated congeners of polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) from their major sources to humans are modeled to estimate the time course, from the past to the future, of the human health risk to Japanese. The modeling approach seems to represent reasonably well the background levels of congeners, except 2,3,7,8-TCDF, in the environment, daily intake and body burden, from the results of a comparison between estimated and measured values. Although PCDD/Fs in herbicides contributed to the past high daily intake and body burden to Japanese, the main sources of the present intake and burden of PCDD/Fs is estimated to be incinerators. The margin of exposure (MOE) for the risk of reproductive alteration in female offspring exposed prenatally was calculated based on the estimated maternal body burden. The MOE values may not be sufficiently large to guarantee the safety of female offspring of mothers born in the 1950s. However, the MOE values for female offspring born in and after the latter half of the 1990s may be sufficiently large to guarantee safety.

Key Words: polychlorinated dibenzo-*p*-dioxin, polychlorinated dibenzofuran, body burden, reproductive alteration, female offspring

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1. Introduction

In order to evaluate the risks caused by chemical substances and to establish appropriate countermeasures for the reduction of the risk when necessary, how precisely and rapidly we assess exposure to the substances through several pathways is crucial. We take chemical substances into our body through the inhalation of air, ingestion of foods, and skin contact with materials containing the substances. Mathematical models are applied to estimate concentrations of substances in environmental media and foodstuff related to the exposure. The models facilitate the estimation of the temporal and/or spatial variation of the concentrations in the environment, foodstuff, and human body, than their measurement. However, the validation of these models is still insufficient, because of the necessity for information regarding the meteorological, environmental, and physiological situations in addition to the concentrations.

Since the latter half of the 1990s, various types of monitoring of dioxins in environmental and biological media have been carried out intensively in Japan, because of the increasing social concern about the risks posed by this chemical group (Environment Agency (EA), 1999a, 1999b, 1999c, 2000; Hori et al., 1999; Iida et al., 1999; Ministry of Health and Welfare (MHW), 1998a, 1999). Furthermore, the Japanese Government has also established several countermeasures such as the reduction of dioxin levels in emissions from incinerators. However, the present daily intake and body burden do not reflect the high-dose exposure to large quantities of dioxins released into the environment as impurities in some herbicides and polychlorinated biphenyls in the past (Masunaga, 1999). Indeed, it has been suggested that the intake of dioxins via foods and the body burden of dioxins in Japanese were much higher in the past, based on the results of a recent analysis of breast milk and foods stored in the past (MHW, 1998a, 1999), although there has been less study of the past dioxin levels in the Japanese environment and in the human body.

Although we have already illustrated the modeling approach to the representation of the transport processes of polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) from their

major sources to the human body in terms of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) toxic equivalents (TEQs) (Yoshida et al., 2000a), seventeen 2,3,7,8-chlorinated congeners assigned nonzero toxic equivalent factors (TEFs) exhibit clear differences in behavior in the environment and the human body (Yoshida et al., 2000b; Yoshida, 2000). Thereto, it has been reported that rather different congener patterns of PCDD/Fs are exhibited among exhaust gas from incinerators, herbicide PCP, and herbicide CNP (Hagenmaier et al., 1994; Masunaga and Nakanishi, 1999).

In this study, we modeled the transport processes of seventeen 2,3,7,8-chlorinated congeners of PCDD/Fs from their major sources to the human body and estimated the time courses of environmental concentrations, daily intake, and body burden of the congeners. These estimates were compared with those reported to confirm the reliability of the model, and then the estimated body burden was applied to the assessment of the risk of reproductive alteration in female offspring exposed prenatally.

2. Description of Modeling Approaches

To estimate the body burden of congeners of PCDD/Fs in the general Japanese population, we considered the sources, environmental transport pathways, and exposure pathways shown in Figure 1. As the emission rate of each congener from incinerators and as herbicide impurities, we used estimates made by Masunaga (Masunaga, 1999; Masunaga and Nakanishi 1999).

1) Congeners emitted from incinerators

The air/soil two-compartment model was applied in order to estimate the environmental concentrations of congeners of PCDD/Fs from incinerators in the air and soil other than that in paddy fields:

$$\frac{dMa}{dt} = E - k_{11} \bullet Ma + k_{12} \bullet Ms$$

$$\frac{dMs}{dt} = k_{21} \bullet Ma - k_{22} \bullet Ms ,$$

where *Ma* and *Ms* are the mass of congeners in air and soil, respectively. *E* is for the emission

rate into air. k_{11} and k_{22} are the lumped 1st-order rate constants for the transport, transfer, and transformation processes of congeners in air and soil, respectively. k_{12} and k_{21} are the lumped 1st-order rate constants for transfer between air and soil, respectively. A detailed description of this model has been presented elsewhere (Yoshida et al., 2000c).

The mass transported from the soil, other than that in paddy fields, to the coast was also calculated using this two-compartment model.

2) Congeners released into paddy fields

The concentrations of the congeners of PCDD/Fs in paddy fields were estimated using the one-compartment model (Yoshida et al., 2000a):

$$\frac{dMpf}{dt} = -kpf_{total} \bullet Mpf \qquad @\ t = 0, \quad Mpf = APPL,$$

where Mpf is the mass of congeners in the soil of paddy fields, and $APPL$ is the mass of the released congeners. We assumed that PCDD/Fs were released to paddy fields once a year. kpf_{total} is the lumped 1st-order rate constant for the removal of congeners in the paddy soil. The volatilization, runoff, erosion and degradation processes contribute to the removal of PCDD/Fs from the top layer of the paddy soil (0-5 cm), but only degradation contributes to the removal from the lower layer of the soil (5-20 cm). The two layers are tilled once a year, and concentrations of PCDD/Fs in the two layers become uniform. The one-compartment model was also applied to calculate the mass transported from the paddy fields to the coast.

3) Congeners in coastal environment

The concentrations of the congeners in the coastal environment were estimated using the water/sediment two-compartment model:

$$\frac{dMw}{dt} = I_{np,sea} + I_{pf,sea} - k_{33} \bullet Mw + k_{34} \bullet Mse$$

$$\frac{dMse}{dt} = k_{43} \bullet Mw - k_{44} \bullet Mse\,,$$

where Mw and Mse are the mass in water and in sediment, respectively. k_{33} and k_{44} are the

lumped 1st-order rate constants for the transport, transfer, and transformation processes of congeners of PCDD/Fs in water and sediment, respectively. k_{34} and k_{43} are the lumped 1st-order rate constants for transfer between water and sediment, respectively. A detailed description of this model has been presented elsewhere (Yoshida et al., 1987).

4) Daily intake of congeners

The daily intake of each congener was individually calculated as products of the inhalation or ingestion rate of air, fish/shellfish, leaf crops, root crops, meat, or dairy products and the concentration of the congener in them, assuming that the ingestion rates of various foods were constant.

The concentration in coastal fish was calculated based on the assumption of equilibrium between the concentrations in water and in fish. The concentrations in offshore and oceanic fish were assumed to be half of that in coastal fish (MHW, 1996). The ratio of ingestion rates of coastal fish and other fish was assumed to be 0.4 and 0.3 before and after 1970, respectively.

The concentrations in leaf/root crops, meat, and dairy products were estimated according to the methods described by the U.S. EPA (1994). We assumed that the epidermis of rice plants was contaminated by PCDD/Fs and residual culms and blades after threshing rice were fed to domestic animals as fodder. The concentrations of congeners in the culm and the blade were calculated based on congener-specific concentrations in irrigated water in paddy fields and the root concentration factors estimated from 1-octanol/water partition coefficients. The utilization ratio of culms and blades as fodder was assumed to decrease from 1.0 in 1958 to 0.1 in 1985 and to be constant thereafter.

5) Body burden and concentration in breast milk

The body burdens of congeners of PCDD/Fs at age t ($C_{human}(t)$) were estimated using the one-compartment model:

$$dC_{human}(t)/dt = \sum_i BA_i \bullet IT_i(t)/BW(t) - ke \bullet C_{human}(t),$$

where subscript *i* stands for intake routes of PCDD/Fs (inhalation of air and ingestion of foods). *BA* is the congener-specific bioavailability, and *IT*(*t*) and *BW*(*t*) are the intake rate and body weight of Japanese at age *t*, respectively. *ke* is the congener-specific 1st-order rate constant for elimination.

The concentration of congeners of PCDD/Fs in breast milk ($C_{milk}(t)$) was calculated as

$$C_{milk}(t) = C_{human}(t)/Fat(t),$$

where *Fat*(*t*) is the fat content at age *t*.

In the calculation, we used congener-specific *BA* and *ke* in humans (Hrudey et al., 1996; Liem and Theelen, 1997) and age-specific *BW*(*t*) and *Fat*(*t*), which are available from the Health and Nutrition Information Infrastructure Database jointly developed by the National Institute of Health and Nutrition and Japan Science and Technology Corporation.

Although we had to employ the body burden as a measure of exposure, as described below, TEFs are based on the administered doses of seventeen 2,3,7,8-chlorinated congeners of PCDD/Fs, not on their tissue doses. We therefore calculated the correction factors for 2,3,7,8-TCDD toxic equivalent body burdens of the seventeen congeners (*CF*), based on the steady-state assumption using congener-specific gastrointestinal absorption (*ABS*) and half-life (*DT*50):

$$CF_i = \frac{TEF_i}{TEF_{TCDD}} \bullet \frac{ABS_{TCDD} \bullet DT50_{TCDD}}{ABS_i \bullet DT50_i},$$

where subscript *i* stands for each congener.

A rounding-off procedure (nearest 1 or 5) was employed to determine the correction factors, because the TEFs are “order of magnitude” estimates of relative toxicity when compared to 2,3,7,8-TCDD (van den Berg et al., 1998). The calculated correction factors for the seventeen congeners are listed in Table 1.

6) Estimation of risk

As the endpoint for the assessment of human health risk, we selected morphological reproductive alterations in female offspring as a consequence of in utero exposure (Gray et al., 1997), because the tolerable daily intake (TDI) of dioxins for Japanese has been determined based on this reproductive toxicity (Central Environment Council/Living Environment Council/Food Sanitation Investigation Council, 1999). Based on data obtained by Hurst et al. (2000), we calculated a 28.6 ng/kg body burden of 2,3,7,8-TCDD between gestation day 16 and 21 for rats administered 2,3,7,8-TCDD at a dosage level of 50 ng/kg, at which Gray et al. found no significant morphological alteration statistically. We described the risk of reproductive alteration as the margin of exposure (MOE). The margin was calculated by dividing the body burden for no-observed-adverse-effect level (28.6 ng/kg) by the estimated maternal body burden.

3. Results

1) Environmental levels of congeners

The estimated time courses of the congener-specific concentrations in the air and the soil of paddy fields are shown in Figure 2 and Figure 3, respectively. A considerable number of calculated congener-specific concentrations in air and soil of paddy fields were in good agreement, within a factor of four, with median values of concentrations measured by the Environment Agency (EA, 1999a, 2000).

2) Daily intake of congeners

Estimated daily intakes of seventeen congeners via food ingestion were compared with those reported (MHW, 1999). As shown in Figure 4, the estimated total daily intakes via food ingestion are in good agreement with those measured, except in 1988. This discrepancy of estimated and measured daily intakes may be due to underestimate of emission rates of CNP-derived PCDD/Fs in 1988. Furthermore, the daily intakes of the major congeners, 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD, and 2,3,4,7,8-PeCDF, are also in good

agreement with those measured. However, the estimated daily intakes of 2,3,7,8-TCDF are one or two orders of magnitude lower than those measured. Figure 5 shows estimated contribution ratios of different sources to daily intakes. As shown in this figure, daily intake of PCDD/Fs is mainly due to their emissions from incinerators after 1992.

3) Concentration in breast milk and body burden of congeners

Figure 6 shows estimated concentrations of 2,3,7,8-TCDD, OCDD, 1,2,3,6,7,8-HxCDF and 1,2,3,4,6,7,8-HpCDF in the fat of 27-year-old females, together with those measured in the breast milk of 25 - 29-year-old females (Hori et al., 1999). As shown in this figure, the tendencies of measured and estimated concentrations toward lower levels of these congeners in fat are in good agreement. These results support the validity to use the congener-specific bioavailability and elimination rate constant. Furthermore, the estimated concentrations of other congeners, except 2,3,7,8-TCDF, are also in reasonable agreement with those measured. The estimated concentrations of 2,3,7,8-TCDF are two orders of magnitude lower than those measured.

Figure 7 shows the time courses of the body burdens of PCDD/Fs in females born in 1950, 1960, 1970 and 1980 in terms of 2,3,7,8-TCDD toxic equivalent body burdens and also contribution ratios of sources to the body burdens. Although PCDD/Fs in PCP and CNP contributed to the high body burdens of Japanese in the past, the incinerators are the main sources of PCDD/Fs at this time, as well as daily intake described above. These results indicate that we must address the time course of daily intake and body burden for assessing reality-based risk to Japanese caused by PCDD/Fs.

4) Estimated risk

Table 2 lists the total toxic equivalent body burden of the PCDD/Fs in 25 - 30-year-old females who were born in 1950, 1960, 1970, and 1980 and the MOE for the risk of reproductive alteration in their female offspring. As shown in this table, the MOE for female offspring of mothers born in 1950 and 1960 were two- or threefold smaller than the MOE for the offspring of

mothers born in 1980, as a consequence of the higher body burden of PCDD/Fs in the mothers of the former.

4. Discussion

In this study, we estimated the congener-specific body burden of PCDD/Fs taking account of the transport processes from sources to humans. The results of the modeling approaches seem to give reasonable estimates of the current and past background levels of congeners of PCDD/Fs in the environment, daily intake, and body burden, except for 2,3,7,8-TCDF. Although the estimated concentrations of 2,3,7,8-TCDF in the air and soil in paddy fields are lower than those measured, they are of the same order of magnitude. However, the estimated daily intakes of this congener via the ingestion of leaf and root crops, fish and shellfish, meat, and dairy products are one or two orders of magnitude lower than those measured. These underestimates are propagated to the body burden estimates. 2,3,7,8-TCDF is a characteristic congener in effluent and sludge from paper manufacturing (Wakimoto, 1991). However, estimated release rates from the bleaching process and the incineration of sludge in paper manufacturing are low (about 6 g-TEQ/year) (Hiraoka and Okajima, 1998). The discrepancy between estimated and measured daily intake and body burden cannot be explained by 2,3,7,8-TCDF from this source. Because we have already validated the parameters describing the transport from an incinerator to the soil through the air, we will have to reassess the main sources of 2,3,7,8-TCDF and parameters describing the transport of this congener from the soil to the human body hereafter, in order to clarify the reason for the discrepancy.

The MOE values were estimated to be 4 - 6 for female offspring of maters born in the 1950s. The margin for the risk of reproductive alteration might not be sufficiently large to guarantee their safety, when we consider that an uncertainty factor of 3 – 10 is applied to extrapolate from the general population to the sensitive subpopulation (Dieter and Konietzka, 1995) and also that the body burden of 2,3,7,8-TCDF was underestimated in our simulation. However, the MOE for

female offspring born in and after the latter half of the 1990s may be sufficient to guarantee safety.

5. Conclusions

In order to establish appropriate countermeasures for the reduction of risk caused by chemical substances in the environment, we must identify the main sources by correlating their emission rates from various sources with descriptors of exposure, such as daily intake and body burden, using mathematical models. The modeling approach allows us to quantitatively compare the effectiveness of more than one countermeasure for reducing the risk level and then to select the most effective one from several alternatives. In the case of dioxins, the reduction of emission from incinerators can be confirmed to be effective for decreasing the human health risk to the general Japanese population in the present and the future, as revealed in this study.

Although modeling approaches have been applied in screening-level risk assessment of organic chemical substances, we must continue to improve the mathematical models for the environment, intake and humans, in order to achieve more reliable estimates of site-specific exposure and risk of many kinds of chemical substances released into the environment, together with the development of databases for exposure factors of human and other organisms.

Acknowledgment

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List of Tables

Figure Captions

Table 1 Correction factors for calculating 2,3,7,8-TCDD toxic equivalent body burden

Congener	Gastrointestinal absorption*	Half-life* (year)	WHO-TEF	Equivalent body burden	Correction factor
2,3,7,8-TCDD	0.97	6.2	1	1.00	1
1,2,3,7,8-PeCDD	0.99	8.6	1	1.42	0.5
1,2,3,4,7,8-HxCDD	0.98	8.4	0.1	13.7	0.05
1,2,3,6,7,8-HxCDD	0.97	13.1**	0.1	21.1	0.05
1,2,3,7,8,9-HxCDD	0.96	8.5	0.1	13.6	0.05
1,2,3,4,6,7,8-HpCDD	0.86	6.6	0.01	94.4	0.01
OCDD	0.76	5.6	0.0001	7080	0.0001
2,3,7,8-TCDF	0.97	0.4	0.1	0.65	1
1,2,3,7,8-PeCDF	0.99	0.9	0.05	2.96	0.5
2,3,4,7,8-PeCDF	0.98	9.9	0.5	3.23	0.5
1,2,3,4,7,8-HxCDF	0.97	5.7	0.1	9.19	0.1
1,2,3,6,7,8-HxCDF	0.97	6.2	0.1	10.0	0.1
1,2,3,7,8,9-HxCDF	0.95	1.1***	0.1	1.74	0.5
2,3,4,6,7,8-HxCDF	0.96	2.4	0.1	3.83	0.5
1,2,3,4,6,7,8-HpCDF	0.87	2.6	0.01	2.89	0.5
1,2,3,4,7,8,9-HpCDF	1.00	0.2****	0.01	53.2	0.01
OCDF	0.95	0.2	0.0001	316	0.005

TCDD : tetrachlorodibenzo-*p*-dioxin
PeCDD : pentachlorodibenzo-*p*-dioxin
HxCDD : hexachlorodibenzo-*p*-dioxin
HpCDD : heptachlorodibenzo-*p*-dioxin
OCDD : octachlorodibenzo-*p*-dioxin
TCDF : tetrachlorodibenzofuran
PeCDF : pentachlorodibenzofuran
HxCDF : hexachlorodibenzofuran
HpCDF : heptachlorodibenzofuran
OCDF : octachlorodibenzofuran

*:Data from Liem and Theelen. (1997).

**: Data from Flesch-Janys et al. (1996)

***: Data from Yoshida (2000).

****: Estimated in this study

Table 2 2,3,7,8-TCDD toxic equivalent body burden and MOE

Maternal female		Female offspring
Year of birth	Toxic equivalent body burden, ng/kg	MOE
1950	5.8 - 6.4	4 - 5
1960	4.7 - 5.8	5 - 6
1970	3.3 - 3.8	8 - 9
1980	2.1 - 2.5	12 - 14

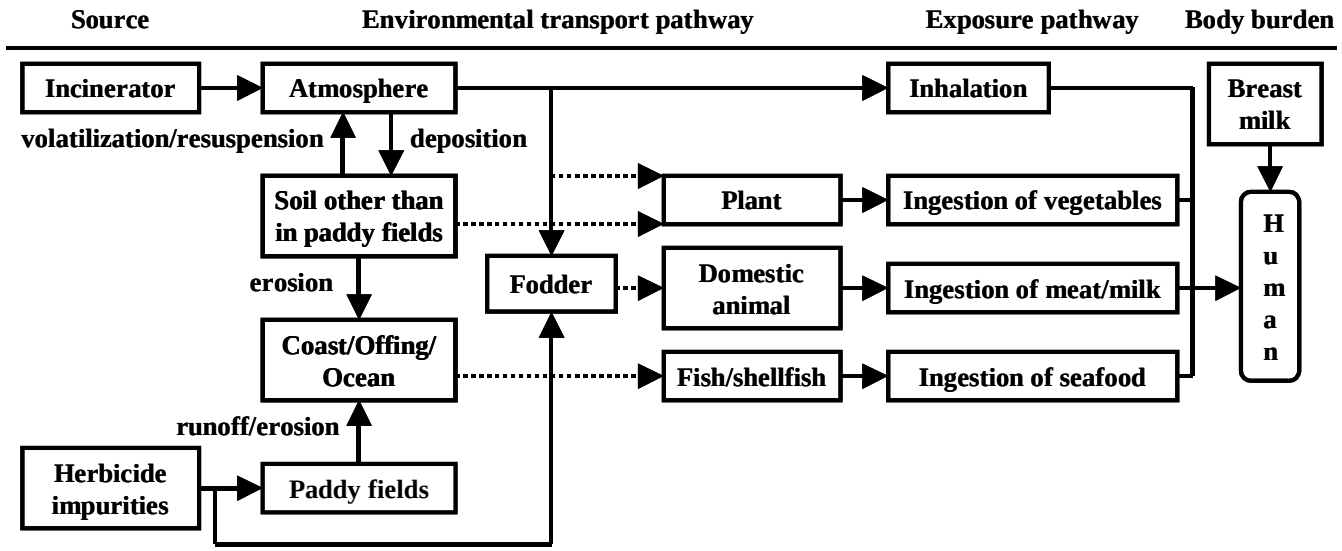

Figure 1 Transport pathways of congeners of PCDD/Fs from sources to human body

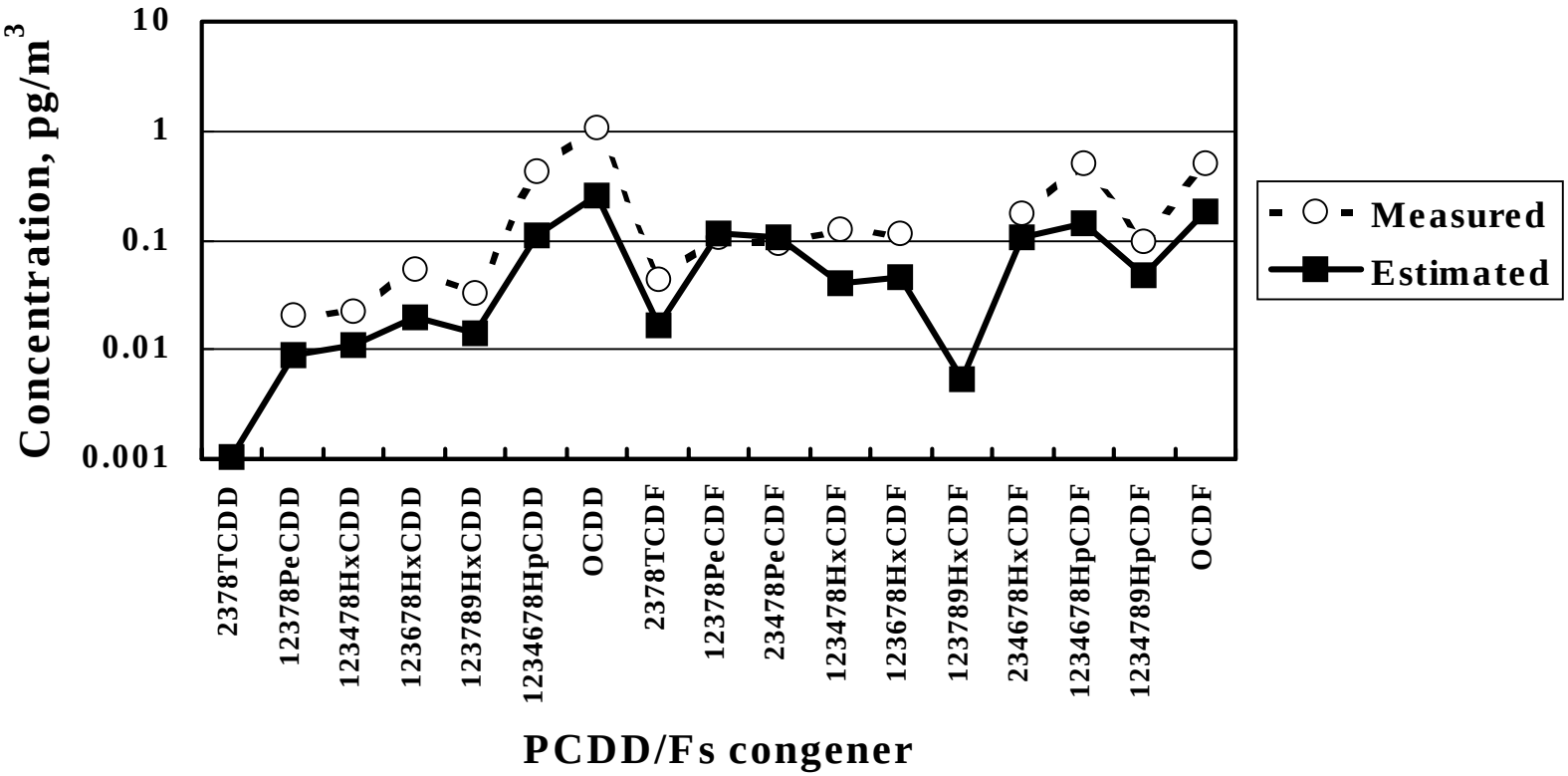

Figure 2 Comparison of measured and estimated congener-specific concentrations in air

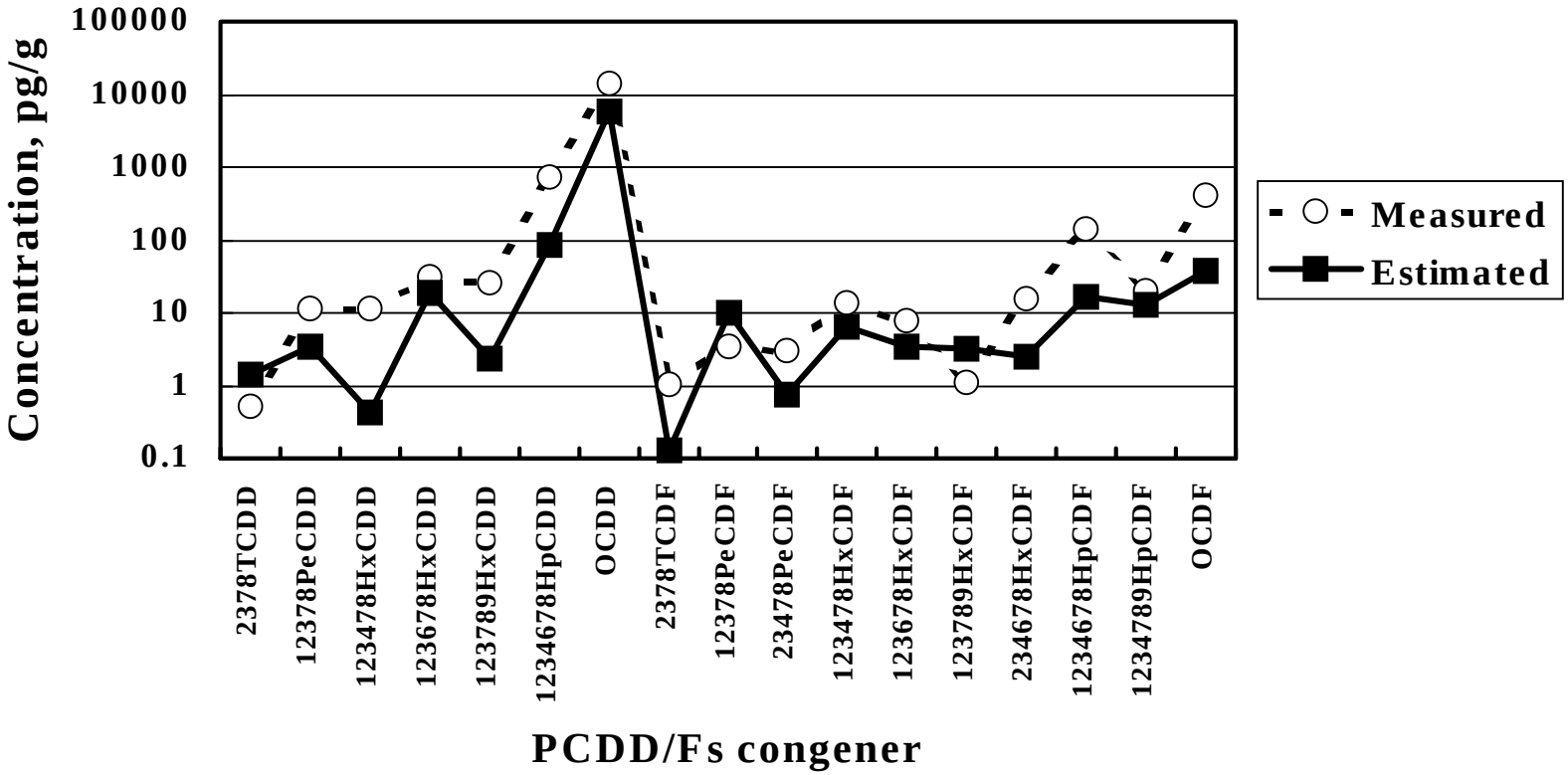

Figure 3 Comparison of measured and estimated congener-specific concentrations in soil of paddy fields

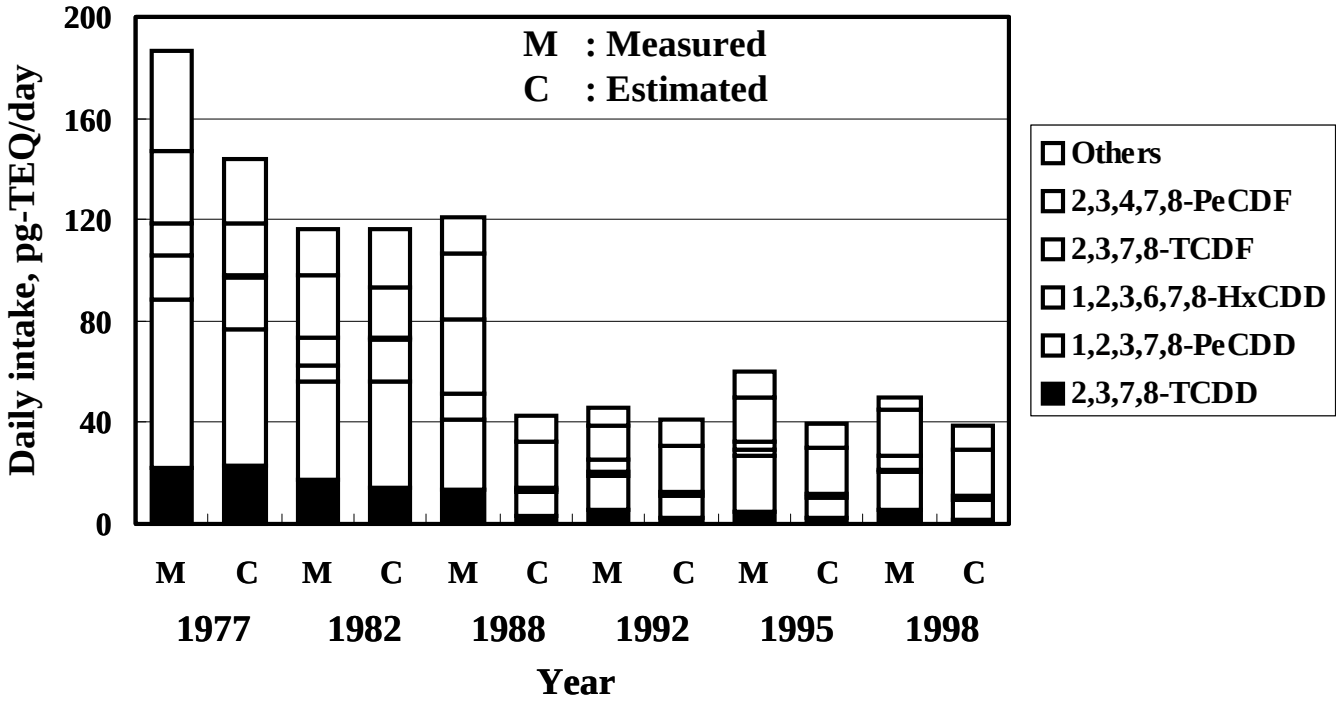

Figure 4 Comparison of measured and estimated daily intakes

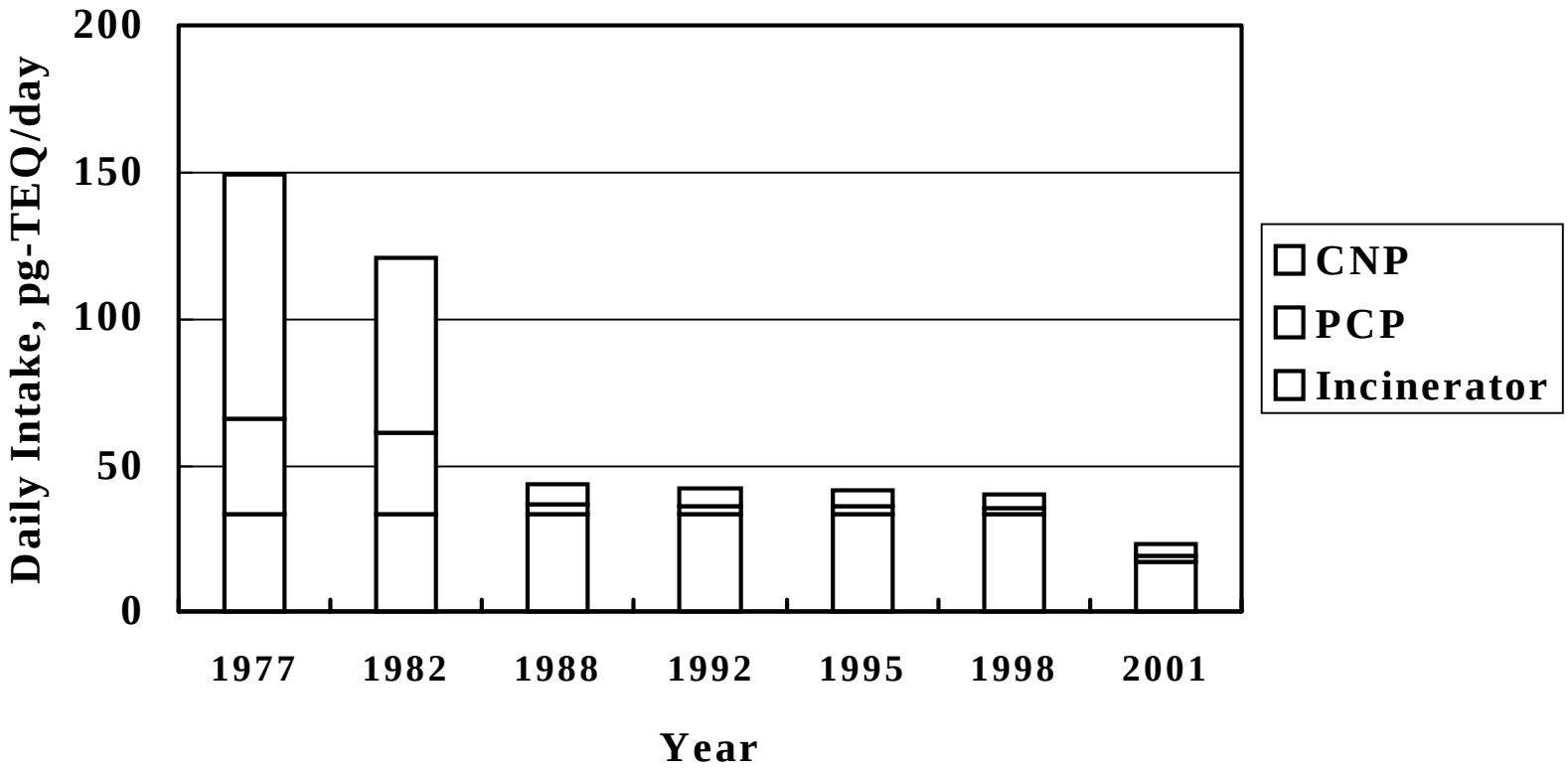

Figure 5 Contribution ratios of different sources to daily intakes

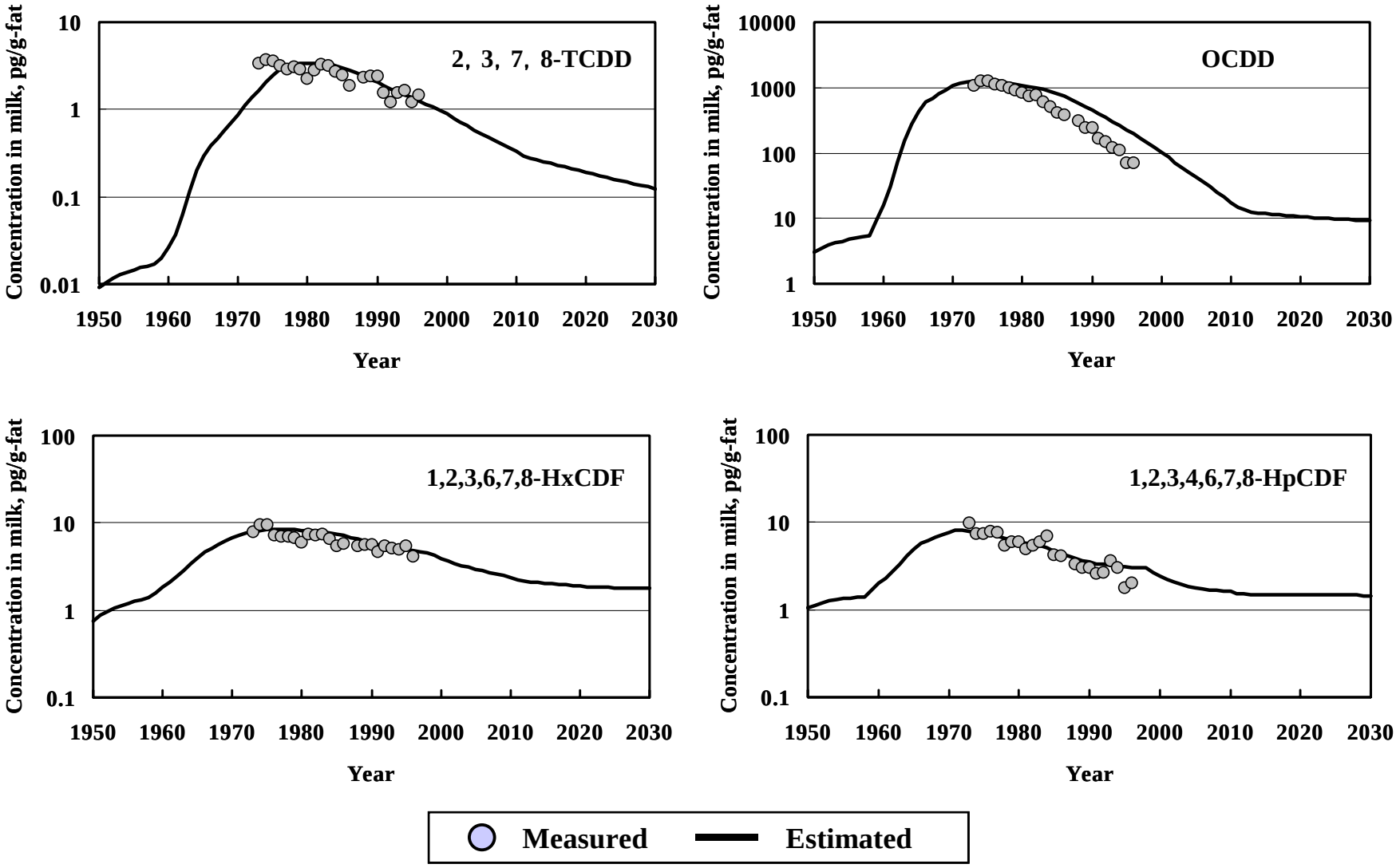

Figure 6 Estimated congener-specific concentrations in the breast milk of 27-year-old females

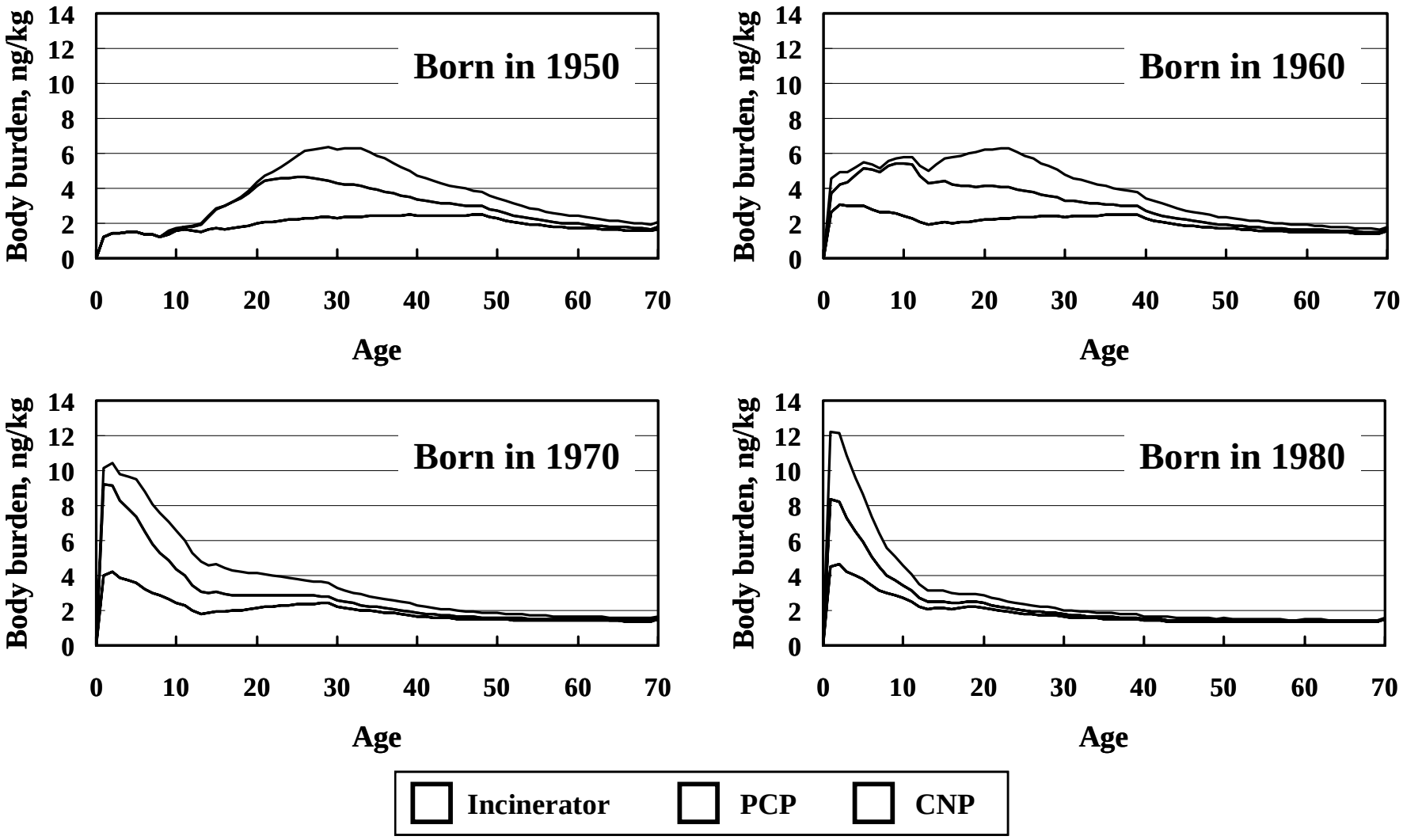

Figure 7 Estimated time courses of toxic equivalent body burdens in Japanese females and contribution ratios of sources