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Original Article

Determination of Dioxins in Human Hair: Estimation of External and Internal Exposure to Dioxins

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Abstract

Objectives: To clarify the origin of dioxin and related compounds (dioxins) in human hair, we determined the amounts of adsorbed dioxins in human hair, and the distribution of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in rats.

Methods: Human hair specimens, packed in a glass column, were exposed to ambient air that was introduced into the column with an air pump for 24 h. Rats were administered TCDD by gavage at doses of 0.2, 0.8, and 1.6 µg/kg body weight. Four weeks after TCDD administration, hair from the back, serum, and adipose tissue were removed under diethyl ether anesthesia. The amounts of dioxins in these samples were analyzed by high resolution gas chromatography with mass spectroscopy.

Results: Exposure of the hair specimens to ambient air for one day increased the total toxic equivalent (TEQ) value by 51%. In TCDD-treated rats, the amount of TCDD in hair increased in a dose-dependent manner, and showed a significant positive correlation with that in adipose tissue.

Conclusions: Human hair was found to retain dioxins by both internal and external exposure, and the contribution of external exposure was estimated to be about 40% of the TEQ.

Key words: dioxins, human hair, accelerated solvent extractor (ASE), absorption, air monitoring

Introduction

Human hair has been used as a sample for non-invasive biomonitoring to estimate the levels of exposure to various chemicals. Hair specimens can be collected repeatedly not only from identical subjects but also from those differing in characteristics, such as age, sex, residential area, food habits, and work environment. Recently, Nakao *et al.* developed a new analytical method for dioxin and related compounds (dioxins) in human hair and showed that the amounts of dioxins in incineration workers' hair were higher than those of the general population (1, 2). Other researchers also used human hair as a bioindicator of exposure to dioxins (3–5). However, to estimate the levels of exposure to dioxins using hair, both the origins of dioxins and the mechanism of their accumulation in human hair remain to be determined.

Dioxins in human hair have been suggested to originate from ambient air based on its congener and isomer profiles (1–5). On the other hand, polychlorinated biphenyls (PCBs) in human hair were reported to reflect the body burden of PCBs (6, 7). Recently, Kitamura *et al.* reported that dioxins were eliminated with sebum from the body (8). These results suggested that dioxins in human hair consist of a mixture of isomers originating from the ambient air and sebum. Therefore, the present study was performed to determine the amounts of dioxins adsorbed not only in human hair, but also in 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD)-treated rat hair, to characterize the origin of dioxins in hair. In addition, we developed and applied a simple analytical method for the rapid and precise determination of dioxins in human hair. Thus, we examined the contribution ratio of internal (sebum) and external (ambient air) exposure to dioxins.

Materials and Methods

Materials

Reagents

TCDD was purchased from Cambridge Isotope Laboratory (Andover, MA, USA). The purity was higher than 99.5%. Nonane and corn oil used to dissolve TCDD or vehicle control

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were from Sigma (St. Louis, MO, USA). ^{13}C-PCDDs/PCDFs and ^{13}C-PCBs as internal standards were from Wellington (Ontario, Canada). Silica gel (Kieselgel 60) and activated carbon impregnated-silica gel were from Merck (Darmstadt, Germany) and Wako Pure Chemical (Osaka, Japan), respectively.

Hair samples

Human hair samples (n=14) were collected from 4 healthy volunteers (male, 3; female, 1; age, 26–33 years old) after washing with shampoo at hairdresser's shops in Tsukuba City and Ryugasaki City, Ibaraki Prefecture, Japan, in 1999–2000. The hair specimens from the volunteers who had no known history of occupational exposure to dioxins were collected after their informed consents. The collected hair samples were less than 5 mm in length, and stored at 4°C until analysis.

Animals

Male and female Holtzman rats were purchased from Harlan Sprague-Dawley, Inc. (Indianapolis, IN, USA) and bred at the animal center of the National Institute for Environmental Studies (NIES). They were maintained under the following conditions: a 12-hr light-dark cycle, a temperature of 23±1°C, and humidity of 50±10%. The animals were given food and water *ad libitum*, and exposed to filtered clean air in separate chambers. Ten-week-old female rats in proestrus were mated 1:1 with males overnight, and females that had a vaginal plug the following morning were designated as being at Day 0 of gestation (9). The rats received humane care throughout the experiment according to the guidelines for animal experiments at the NIES.

Sample treatment and analysis

Development of new method for determination of dioxins in human hair

The hair specimen from one person was divided into two portions. One portion was treated according to the method described by Nakao *et al.* (1). Briefly, the hair specimen, which was further divided into three subportions of ca. 2.0 g each, was cut into pieces less than 1 mm in length, and spiked with ^{13}C-PCDDs/PCDFs as an internal standard, followed by digestion in 2.0 M potassium hydroxide solution (20 ml) for 12 h. The digested materials were washed into a separation funnel with an identical volume (20 ml) of methanol, from which dioxins were extracted with *n*-hexane. The *n*-hexane layer was cleaned up using concentrated sulfuric acid. The solution was concentrated and cleaned up further by silica gel and activated carbon-impregnated silica gel column chromatography. The other portions of the hair specimens, which were also divided into 3 subportions of ca. 2.0 g, were extracted using an accelerated solvent extractor (ASE-200, Dionex Co., Sunnyvale, CA, USA) as described below. Solvent, *n*-hexane/acetone (50/50, v/v); temperature, 150°C; pressure, 13.8×10^6 Pa (2,000 psi); heat, 7 min; static time, 5 min; flush volume, 80%; purge time, 60 sec; cycle, 3 times. The extract was concentrated on a rotary evaporator under reduced pressure. Then, the extract was digested in 2.0 M potassium hydroxide with internal standard, and cleaned up in the same manner as described above.

Adsorption of dioxins on hair and wool

The hair specimen from one person was exposed to ambient air as described below. Hair specimens (ca. 2.0 g, n=3) were packed into a glass column (10 mm i.d., ×80 mm in length), and exposed to ambient air with an air pump for 24 h (rate, 20 L/min) at the roof of the NIES building.

Commercial wool was also exposed to ambient air without ventilation using an air pump. Balls of wool (ca. 2.0 g, n=3) were hung in a room or outdoors in Tsukuba City for 3 months. These hair and wool specimens were extracted with ASE-200 and cleaned up in the same manner as described above.

Administration of TCDD in pregnant Holtzman rats

On gestation day 15 (GD15) pregnant Holtzman rats were administered TCDD by gavage at doses of 0.2, 0.8, and 1.6 μg/kg body weight in corn oil (2.5 ml/kg). Four weeks after TCDD administration, dams were sacrificed under diethyl ether anesthesia to collect visceral adipose tissue, serum, and hair specimens from the back, and these specimens were stored at −20°C until analysis (9). On day 135 after birth, the offspring of dams administered TCDD at a dose of 0.2 μg/kg body weight on GD15 were also sacrificed to collect adipose tissue and hair. Rat hair specimens were subjected to extraction of dioxins with ASE-200, and the extract was digested and cleaned up as described above. Serum and adipose tissue specimens were also digested and cleaned up for TCDD quantification.

Quantification of Dioxins

High resolution gas chromatography and high resolution mass spectroscopy (GC/MS) were performed in the selected ion mode on a JMS-700 high-resolution double-focusing mass spectrometer (JEOL, Tokyo, Japan) coupled to an HP 6890 gas chromatograph (Hewlett Packard, Wilmington, DE, USA). The sample solution was introduced into an HP 6890 equipped with a CP-SIL 8CB/MS column (Chrompack, EA Middelburg, Netherlands; 30 m×0.25 mm i.d., film thickness 0.25 μm). A mass resolution of m/Δm >10,000 was used in the EI mode. Identification was based on the correct isotope ratio of M^+ to $(M+2)^+$ (±15%), recoveries (50–120%), and retention times (±4.0 sec) of GC separation. The area of mass profile peaks of the quantification ions was used for quantitative analysis of dioxins. Quantified values were calculated by the internal standard method (9, 10).

Statistical analysis

Data are expressed as means±SD. Differences between mean values were analyzed by Student's *t*-test, and $p<0.05$ was regarded as statistically significant.

Results

Development of simple analytical method for dioxins in human hair

Comparison of the analytical method

Nakao's method (1) is time-consuming because hair speci-

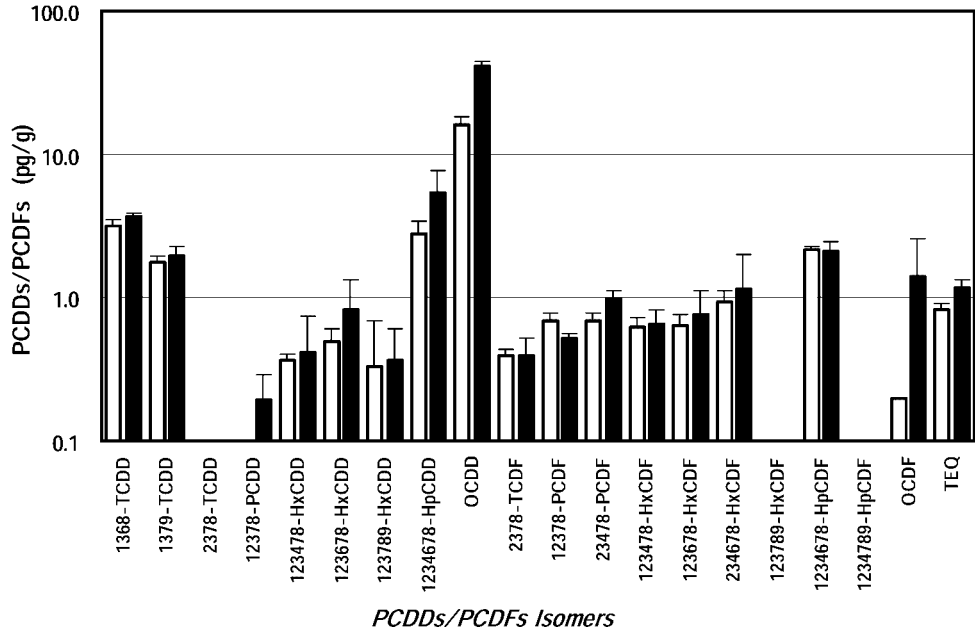

Fig. 1 Comparison of ALK (open bar) and ASE (closed bar) methods on the detected amounts of PCDDs/PCDFs isomers from human hair.

Table 1 Concentration of PCDDs and PCDFs in human hair from four volunteers (Average±SD pg/g)

Isomers	Samples			
	Volunteer 1 (n=6)	Volunteer 2 (n=2)	Volunteer 3 (n=3)	Volunteer 4 (n=3)
1,3,6,8-TeCDD	3.91±1.33	3.52	2.34±0.80	4.30±0.57
1,3,7,9-TeCDD	1.86±0.69	1.66	1.10±0.46	2.00±0.32
2,3,7,8-TeCDD	0.11±0.08	0.14	0.15±0.08	0.09±0.03
1,2,3,7,8-PeCDD	0.27±0.12	0.46	0.26±0.11	0.31±0.18
1,2,3,4,7,8-HxCDD	0.19±0.06	0.18	0.17±0.10	0.15±0.03
1,2,3,6,7,8-HxCDD	0.46±0.15	0.74	0.44±0.18	0.52±0.30
1,2,3,7,8,9-HxCDD	0.24±0.11	0.32	0.17±0.03	0.35±0.18
1,2,3,4,6,7,8-HpCDD	2.97±1.26	3.12	1.96±1.39	3.43±0.56
OCDD	28.9±8.66	43.0	12.4±3.54	35.3±8.13
2,3,7,8-TeCDF	0.45±0.05	0.38	0.52±0.25	0.47±0.10
1,2,3,7,8-PeCDF	0.46±0.13	0.28	0.58±0.28	0.31±0.04
2,3,4,7,8-PeCDF	0.95±0.30	1.08	0.92±0.15	0.79±0.31
1,2,3,4,7,8-HxCDF	0.46±0.15	0.39	0.59±0.15	0.40±0.18
1,2,3,6,7,8-HxCDF	0.49±0.14	0.38	0.58±0.13	0.44±0.15
2,3,4,6,7,8-HxCDF	0.57±0.20	0.53	0.46±0.09	0.61±0.19
1,2,3,7,8,9-HxCDF	0.05±0.04	0.07	0.02±0.03	0.10±0.06
1,2,3,4,6,7,8-HpCDF	1.46±0.40	1.62	1.22±0.39	1.63±0.51
1,2,3,4,7,8,9-HpCDF	0.13±0.06	0.11	0.03±0.04	0.16±0.08
OCDF	1.18±0.84	0.68	0.18±0.20	0.72±0.23
TEQ (pg/g-dry)	1.08±0.32	1.32	1.10±0.20	1.05±0.25
Lipid (%)	*5.37±1.59*	*4.55*	*11.8±0.10*	*2.51±0.21*
TEQ (pg/g-lipid)	*22.1±10.9*	*28.8*	*9.35±1.78*	*42.6±13.6*

n: sample number.

mens must be cut into pieces less than 1 mm in length to be digested readily in alkaline solution, whereas others have reported that the use of ASE-200 for extraction of dioxins from animal organs resulted in high recovery in a short time (11, 12). Thus, we modified Nakao's method by the addition of a lipid extraction step using ASE for extraction of dioxins from human hair specimens before alkaline digestion. In comparison with Nakao's direct alkaline digestion (ALK) method, the modified method with ASE extraction and alkaline digestion (ASE) yielded higher levels of dioxins (Fig. 1). Particularly, the amounts of hepta- and octa-chlorinated isomers were significantly increased, and the TEQ, calculated from the WHO toxicity equivalency factors (TEF) (13), was increased by 30% in our modified method.

Application of the ASE method to human hair

The amounts of dioxins in human hair from four volunteers are summarized in Table 1. The amounts of dioxins were very similar in all of these subjects (1.05–1.32 pg-TEQ/g) on a dry weight basis, whereas the amount on a lipid basis was different among the volunteers (9.4–42.6 pg-TEQ/g-lipid). The hair specimens contained less-toxic dioxin isomers, such as 1,3,6,8-tetrachlorodibenzo-*p*-dioxin, which were not given TEFs similarly to the environmental samples (14).

Adsorption of dioxins on human hair and wool

The amounts of dioxins in specimens of human hair were compared before and after exposure to ambient air for specified periods, and the results are summarized in Table 2. Exposure of the hair specimens to ambient air for one day increased the total TEQ by 51%. Marked increases in the adsorbed amounts were observed particularly with regard to less-chlorinated isomers of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and coplanar polychlorinated biphenyls (Co-PCBs), presumably affected by the vapor pressure of each isomer. In addition, commercial wool specimens were found to adsorb dioxins from ambient air. The TEQ (PCDDs+PCDFs) in the blank wool was very low (0.11±0.09 pg/g), whereas exposure to room or ambient air for 3 months increased the levels of dioxins to 1.37–2.74 pg-TEQ/g-dry or 5.65–13.0 pg-TEQ/g-dry, respectively. The exposed wool contained less-toxic isomers, such as 1,3,6,8- and 1,3,7,9-tetrachlorodibenzo-*p*-dioxins. The isomer profile of dioxins in the wool specimens was similar to that of human hair specimens, 1,3,6,8- and 1,3,7,9-tetrachlorodibenzo-*p*-dioxins, 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin and octachlorodibenzo-*p*-dioxin were predominant isomers in both samples.

Distribution of dioxins in the rat

To evaluate the transfer of TCDD from the body to the hair, rats were administered TCDD, which resulted in an

Table 2 Adsorption of dioxins on normal Japanese hair from ambient air (Average±SD pg/g-dry)

Isomers	Samples			
	Blank (n=3)	Exposure (n=3)	Adsorption (pg/g)	% of increase
1,3,6,8-TeCDD	4.08±0.87	9.29±2.65	5.21	*127.7*
1,3,7,9-TeCDD	1.78±0.35	3.86±1.18	2.08	*116.9*
2,3,7,8-TeCDD	<0.44	<0.60	—*	—
1,2,3,7,8-PeCDD	0.19±0.02	0.31±0.05	0.11	*57.9*
1,2,3,4,7,8-HxCDD	<0.44	<0.60	—	—
1,2,3,6,7,8-HxCDD	1.16±0.66	1.57±0.48	0.42	*36.2*
1,2,3,7,8,9-HxCDD	<0.44	<0.60	—	—
1,2,3,4,6,7,8-HpCDD	3.13±0.40	3.70±0.40	0.58	*18.5*
OCDD	22.7±3.63	28.8±4.22	6.10	*26.9*
2,3,7,8-TeCDF	0.36±0.08	0.80±0.17	0.44	*122.2*
1,2,3,7,8-PeCDF	0.18±0.05	0.36±0.15	0.18	*100.0*
2,3,4,7,8-PeCDF	0.45±0.02	0.81±0.06	0.36	*80.0*
1,2,3,4,7,8-HxCDF	0.55±0.39	1.14±0.14	0.59	*107.3*
1,2,3,6,7,8-HxCDF	0.93±0.11	1.44±0.15	0.51	*54.8*
2,3,4,6,7,8-HxCDF	0.53±0.36	1.01±0.17	0.48	*90.6*
1,2,3,7,8,9-HxCDF	<0.44	<0.60	—	—
1,2,3,4,6,7,8-HpCDF	1.48±0.33	2.44±0.62	0.96	*64.9*
1,2,3,4,7,8,9-HpCDF	<0.44	<0.60	—	—
OCDF	1.31±0.70	1.17±0.59	–0.14	*–10.7*
TEQ (PCDDs/PCDFs)	0.30±0.03	0.56±0.04	0.26	*86.7*
3,4,4',5-TeCB	0.45±0.20	0.79±0.19	0.33	*73.3*
3,3',4,4'-TeCB	8.75±1.68	13.4±2.22	4.67	*53.4*
2'3,4,4',5-PeCB	6.29±2.91	10.3±1.11	3.98	*63.3*
2,3',4,4',5-PeCB	158±20.9	181±10.0	23.3	*14.7*
2,3,4,4',5'-PeCB	5.29±1.40	6.74±1.03	1.46	*27.6*
2,3,3',4,4'-PeCB	41.8±7.38	52.4±3.02	10.6	*25.4*
3,3',4,4',5-PeCB	0.85±0.36	1.39±0.34	0.54	*63.5*
2,3',4,4',5,5'-HxCB	11.2±2.62	15.9±3.09	4.70	*42.0*
2,3,3',4,4',5-HxCB	34.6±10.4	34.2±5.25	–0.42	*–1.2*
2,3,3',4,4',5'-HxCB	9.61±2.84	12.0±2.52	2.39	*24.9*
3,3',4,4',5,5'-HxCB	0.49±0.18	0.59±0.14	0.11	*22.4*
2,3,3',4,4',5,5'-HpCB	2.95±0.83	2.22±0.81	–0.73	*–24.7*
TEQ (Co-PCBs)	0.07±0.02	0.20±0.04	0.12	*171.4*
Total TEQ	0.37±0.04	0.75±0.07	0.38	102.7

Human hair was exposed to 14.23±0.93 m^3 of ambient air/g–hair. *: no data.

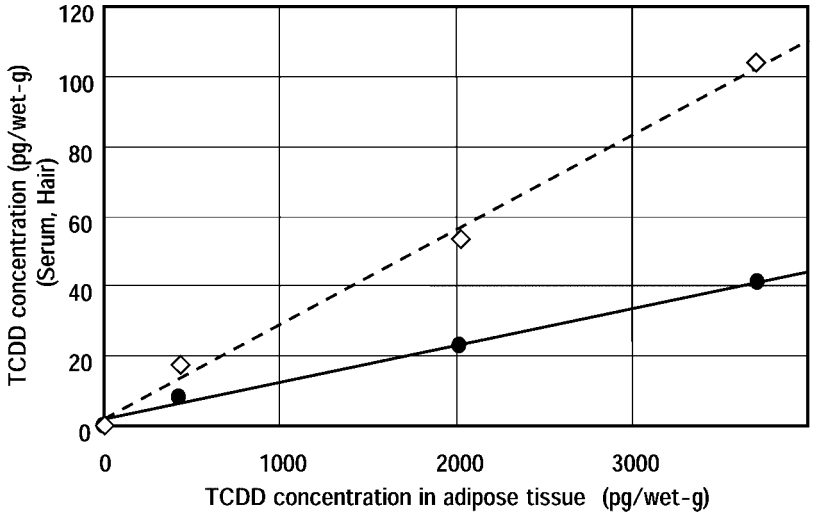

Fig. 2 Correlation of TCDD concentrations in adipose tissue with serum (open rhombus) and adipose tissue with hair (closed circle) of Holtzman rats.

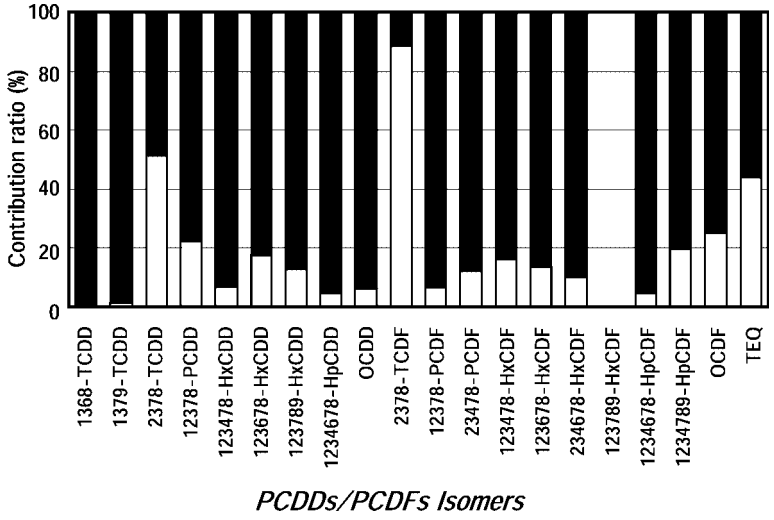

Fig. 3 Contribution ratio of internal (adipose: open bar) and external (air: closed bar) dioxins exposure on the amount of dioxins in rat hair.

increase in the amount of TCDD in the hair in a dose-dependent manner. The amount of TCDD in the rat hair and serum was correlated with those in adipose tissues (Fig. 2). To examine the transfer of dioxins from sebum to hair, we determined dioxins in human sebum and hair from a volunteer (sebum and hair specimens; n=3). Although we could not determine the isomers of PCDDs and PCDFs, the isomer profiles of Co-PCBs in the hair and sebum (data not shown) were determined. The profiles was similar to each other with the exception of 3,3',4,4'-tetrachlorobiphenyl (PCB77), and 2,3',4,4',5 and 2,3,3',4,4'-pentachlorobiphenyls (PCB118, 105) and 2,3,3',4,4',5-hexachlorobiphenyl (PCB156) were predominant isomers in both samples.

Estimation of internal and external dioxin exposure

The isomer profile of dioxins in environmental samples was generally used to estimate the origin of dioxins. As isomer profiles were different between animal and environmental samples, we could not estimate the contribution ratio of internal exposure to external dioxin exposure. We assumed that hair lipid originated from sebum, and that the amounts of dioxins in adipose tissue and sebum were nearly identical on a lipid basis. The amounts of dioxins in the organs have been shown to depend upon the lipid content of a given tissue (15, 16).

Amounts of dioxins of internal and external origins in the hair were calculated for each isomer as follows:

Internal dioxins (pg)=lipid contents in hair (g-lipid/g)×dioxins concentration in adipose tissue (pg/g-lipid)×hair weight (g).

External dioxins (pg)=Total dioxins in hair (pg)–Internal dioxins (pg).

First, we calculated the dioxins of both origins in the hair of rat offspring at PND135 from TCDD-administered dams (200 ng/kg-body weight) using the amounts of each isomer in adipose tissue and hair. The amounts of dioxins in the rat

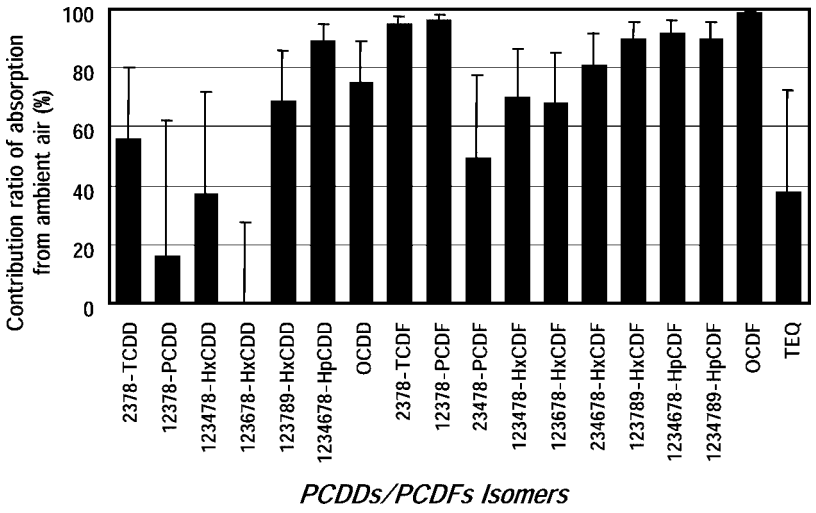

Fig. 4 Contribution ratio of external dioxins exposure on the basis of lipid contents in human hair.

adipose tissue and hair were 19.3 and 43.6 pg-TEQ/g-lipid, respectively, which were similar to the normal human specimens on a lipid basis (17). The percentages of internal and external origins in the rat hair are shown for each isomer in Figure 3. With regard to the origin of isomers, 50% of TCDD and 90% of 2,3,7,8-TCDF were attributed to internal lipid, but other TEF-assigned isomers (PCDDs and PCDFs) were thought to be derived from external adsorption, and about 60% of TEQ was of external origin.

Second, we examined the externally originating dioxins in human hair using our results and the data of dioxin contents in adipose tissue in Japanese subjects with no known excessive exposure to dioxins (17). The percentage (average±SD, n=4) of external contribution was calculated as described above, and shown in Figure 4. The external contribution of each isomer was higher than 50% except for 3 isomers (1,2,3,7,8-pentachlorodibenzo-*p*-dioxin, 1,2,3,4,7,8- and 1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxins), and 40% of TEQ in human hair was thought to be of external origin.

Discussion

In the present study, we developed a simple and high recovery method for dioxin analysis of human hair, and studied the usefulness of human hair as a biomarker of dioxin exposure. The former objective was achieved using ASE to extract hair lipids containing dioxins and by digesting the lipids with alkaline solution. The recovery of dioxins from human hair was improved by this ASE process. In the lipid extraction process, dioxins are thought to be extracted completely with high pressure and high temperature solvents. Substantial amounts of dioxins were not extracted in the ALK method because dioxins are adsorbed tightly with hair protein or airborne particles, and these matrices were not digested with alkaline solution. In addition, our ASE method could save both solvent and time for extraction of dioxins from hair as compared with the Soxhlet extractor widely used. Although our modified ASE method has an additional step as compared to Nakao's ALK method, the former has advantages over the latter in terms of the high recovery of dioxins, faster extraction time (less than 30 min per sample), and the capability to determine lipid content. Thus, our modified ASE method is better than the ALK method and could be applied to determine dioxins in human hair specimens.

The latter objective was achieved by determination of internal and external exposure levels of dioxins in hair based on the following three findings. First, both human hair and wool were found to adsorb dioxins from ambient air, and the external exposure was estimated by the adsorption of dioxins from ambient air to human hair or commercial wool. For environmental monitoring, pine needles have been used as a marker for ambient air concentrations of dioxins (2, 18, 19), but human hair would be more useful for estimation of dioxin exposure level on an individual basis. No changes were found in the levels of TCDD in rat hair specimens following washing with detergent in the present study (data not shown), whereas other researchers reported decreases in the amount of dioxin in human hair during hair washing with shampoo for the first wash, but not for the second (1, 2). This observation regarding the effect of shampoo on human hair is consistent with the results of the present study because the hair specimens obtained at a hairdresser's shop were collected after washing with shampoo. The above results suggest that hair washing had some effect on the amounts of dioxins in hair but could not remove the dioxins from the hair completely. Therefore, the method of washing of human hair must be standardized for dioxin monitoring.

Second, in TCDD-treated rats, the concentration of TCDD in hair and serum was correlated well with those in adipose tissue, suggesting that TCDD was excreted with sebum and that the amount of TCDD in rat hair reflected internal TCDD body burden. The internal exposure was estimated as the transfer of dioxins *via* lipid. The distributions of dioxins in various tissues from laboratory animals have been documented, but the present study is the first report of the amounts of dioxins in TCDD-treated animal hair. We considered that TCDD in the animal hair originated from within the body *via* lipid, and was not adsorption from the ambient air. This hypothesis was supported by the observation that human hair and sebum have very similar

Co-PCB isomer profiles, with the exception of PCB77. On the other hand, it was reported that PCB77, the amount of which is greater in hair than sebum, originates from combustion (20). In a previous study, Kitamura *et al.* reported that dioxins were excreted in human sebum (8), and it is reasonable to speculate that dioxins are transferred to the hair *via* sebum, and to propose that human hair is a good non-invasive biomarker that reflects the dioxin burden in the human body.

As described above, the concentration of dioxins in human hair reflects both internal and external exposure to dioxins on an individual basis. To estimate the level of dioxin exposed on an individual basis, it is necessary to determine the relative contributions of dioxins of internal and external origin in human hair by comparison of isomer profiles between sebum and hair. However, we could not perform direct comparison of the isomer profiles between the two components. Although hair had an isomer composition nearly identical to that of the ambient air except for TCDD, the latter had a larger variety of isomers as compared to biological tissues, including the hair, in which a limited number of isomers are predominantly found.

Third, we determined the tissue concentrations of dioxins in the rat as well as the dioxin isomer profiles in human hair and sebum, and the contribution ratio of internal and external of dioxin exposure levels in hair was estimated on an isomer basis. We assumed that hair lipid originated from sebum and that the amounts of dioxins in adipose tissue and sebum were identical to each other on a lipid basis because the distribution of dioxins is dependent on the lipid content of each tissue or organ. To our knowledge, this is the first report showing the percentages of dioxin isomers of internal and external origin in the hair. The estimated internal exposure levels were about 40% and 60% of TEQs (PCDDs and PCDFs) in rat and human hair, respectively. These estimates were supported by the previous finding that dioxins were excreted with sebum from the skin (8). On the other hand, with the exception of PCB77 (17%), the internal percentage of Co-PCB isomers calculated on an isomer basis in human hair and sebum was approximately 100%. The contribution of internal exposure to the levels of Co-PCBs in human hair was greater than those of PCDDs and PCDFs, suggesting that Co-PCBs in human hair were mainly derived from food, but that PCDDs and PCDFs were from the ambient air as formed in combustion.

In summary, the present study provided a new method of determining the origin and the levels of internal and external dioxin exposure based on a modified method for the extraction of dioxins from human hair specimens. Human hair has the capacity to adsorb dioxins from the ambient air, and the level of TCDD in rat hair was correlated with that in adipose tissue. Human hair is a useful biomarker for chemical exposure, and the levels of dioxins in human hair reflect both ambient air and body burden. We conclude that human hair reflects both internal and external exposure to dioxins and that the levels of dioxins of internal origin accounted for 40–60% of TEQs in the hair of both rats and humans on a lipid basis.

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