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Potential Health Risk via Inhalation/Ingestion Exposure to Polychlorinated Dibenzo-p-dioxins and Dibenzofurans

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Risk assessment and management of polychlorinated dibenzop-dioxins(PCDDs) and dibenzofurans(PCDFs) are being conducted in various countries(U.S. EPA 1987, 1989) because of their highly toxic effects (Kociba et al. 1976). It has been demonstrated that the main route of PCDD/F intake into the human body is by daily ingestion of foods, accounting for approximately 98 % versus all sources and routes of PCDD/F exposure(Travis and Hattemer-Frey 1990). The measured average daily intake as 2, 3,7,8-TCDD toxic equivalents via food consumption is about 1 pg/kg/day as the background level of PCDDs/Fs to which the general population is exposed(Beck et al. 1989; Birmingham et al. 1989; Ono et al. 1987; Roberts 1991). It has been suggested that the tolerable daily intake of 2,3,7,8-TCDD spans a range from 1-10 pg/kg/day(Barnes 1989; Hiremath et al. 1986). We report here the PCDD/F daily intake and health risk by inhalation in the external environment as compared to those for the ingestion exposure to PCDDs/Fs via food consumption.

RESULTS AND DISCUSSION

The long-term health risk via smoking/inhalation or ingestion exposure to PCDDs/Fs are shown in Table 1. 2, 3,7,8-TCDD equivalents by the measured concentrations of PCDDs/Fs in smoke and emission were 1.81 ng/m^3 for

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cigarette smoke(PCDD alone) from laboratory combustion experiments(Muto and Takizawa 1989), 29 ng/m^3 for rice straw smoke from burning experiments(Muto et al. unpublished data), and 2.94 ng/m^3 for emission from incinerating waste chemical oil from an agricultural manufacturer(Muto et al. 1991). These equivalents agree with prior reports by Siebert et al. (1987). The maximum equivalents at ground level of rice straw smoke and of incinerator emission concentrations were recalculated as approximately 5.8 and 0.023 pg/m^3, assuming the diffusion coefficient quoted by the U.S. EPA (1983). For cigarette smoke equivalent for the risk calculation, the reported value 1.81 ng/m^3 was used in order to be inhaled directly into the lung without diffusion and/or dilution. Then, the long-term cancer risk using the U.S. EPA's data for cancer potency (U.S. EPA 1985) by ingestion of daily foods was calculated as 144×10^{-6}, which represented two to three orders of magnitude higher risk than by inhalation reported here. The estimate of incremental PCDD cancer risk from smoking 20 cigarettes/day was higher than those from other inhalation routes.

The measured total daily intake of 2,3,7,8-TCDD equivalents via daily food consumption ranged from 63.0 to 93.5 pg/day (Beck et al. 1989; Birmingham et al. 1989; Ono et al. 1987) as calculated from the concentrations of PCDD/F analogues and using the toxic equivalent factors. The total intake per kg body weight spanned a range from 1.05-1.57 pg/kg/day for a 60 kg adult. It has been suggested that the 2,3,7,8-TCDD equivalents from fatty foods such as meat, milk, fish, eggs and their products make a greater contribution to the PCDD/F daily intake as compared to vegetables and fruit which are eaten in greater amounts (Beck et al. 1989; Birmingham et al. 1989). On the other hand, the calculated total daily intakes of 2,3,7,8-TCDD were 40.5 and 29 pg/day(Travis and Hattemer-Frey 1990) as simulated by using the level-III fugacity model and one compartment pharmacokinetic model, respectively. These calcu-

Table 1. Long-term health risk via smoking/inhalation or ingestion exposure to PCDDs/Fs

Description	Cigarette smoke (smoker)	Rice straw smoke (farmer)	Emission from waste incin. (general popul)	Foodstuffs (general population)	Units
Smoke/emission concentration(SC)	1,810[b]	5.8[c]	0.023[d]		pg/m^3
Smoking/inhalation rate(SR)	$3.5x10^{-3}$[f]	0.83[g]	6.67[h]		m^3/day
Daily intake(DI=SCxSR)	6.34	4.8	0.15	63.0[e]	pg/day
Absorption rate(AB)	0.75	0.75	0.75[i]	0.88[j]	-
Daily intake/body weight(DB=DIxAB/BW)	0.08	0.06	$1.88x10^{-3}$	0.92	pg/kg/day
Exposure duration(ED)	18,250[k]	750[l]	25,550	25,550	days
Incremental cancer risk[a] ($x10^{-6}$)	8.82	0.27	0.29	144.	

[a]Using the upper-limit estimate of incremental cancer risk = DBxEDxl/LFxCPxCF(U.S. EPA 1985), where the cancer potency(CP) and conversion factor(CF) are 156,000$(mg/kg.day)^{-1}$ and 1 $x10^{-9}$ mg/pg, respectively, assuming 60 kg adult body weight(BW) and 70 years of lifetime(LF). [b]Equivalent calculated from total PCDD concentration in cigarette smoke(Muto and Takizawa 1989), [c]Rice straw smoke widely occurs from the paddy fields in the burning season of September to October in Japan ("INAWARA" smog). PCDD/F equivalent in straw smoke, using the international toxic equivalent factor(U.S.EPA 1989), was 29 ng/m^3 at the maximum level from laboratory straw burning experiments(Muto et al.1989: unpublished data), and the maximum equivalent at the ground level is 5.8 pg/m^3, assuming a diffusion coefficient of 5,000. [d]Maximum equivalent at the ground level, using 2.94 ng/m^3 of emission equivalent of PCDDs/Fs, is 0.023 pg/m^3, assuming a diffusion coefficient of 13,000(U.S. EPA 1983). [e]Equivalent calculated from PCDD/F concentrations detected in Japanese foodstuffs and their food consumption rates(Ono et al. 1987). [f]Assuming the international smoking mode as follows: 1 puff/min;inhaled volume/1 puff, 35 ml/2 sec;length of cigarette end, 30 mm. Then, the puff number per one cigarette is approximately 5, and total inhaled volume by smoking 20 cigarettes/day is 3.5×10^{-3} m^3/day. [g]Assumption: 1 hr exposure/farmer/day and 20 m^3/day of inhaled volume(U.S. EPA 1983). [h]Assumption: 8 hr exposure/person/day and 20 m^3 inhal./day. [i]Assuming that 75 % of the particles present in emission are reabsorbed (U.S. EPA 1983). [j]Poiger and Schlatter 1986. [k]50 years(more than 20 year-old), [l]Assumption: 50 years as 15 days/year.

lated intakes almost agreed with the above measured results.

Because of the difficulty of attributing the observed effects solely to the presence of 2,3,7,8-TCDD, the human evidence for the carcinogenicity of 2,3,7,8-TCDD alone is regarded as "inadequate" using either the U.S. EPA or IARC classification criteria (Hiremath et al. 1986). In addition, from the cohort studies on general populations in which the agricultural chemicals including PCDD/F impurities were sprayed, the increases in infant mortalities, various malformations and neoplastic changes can not be statistically attributable to PCDD/F alone(Dai et al. 1990). However, it has been indicated that 2,3,7,8-TCDD can act as a tumor promoting compound(Pitot et al. 1980). Recently, from an epidemiologic study examining the mortality records of essentially all U.S. chemical workers exposed to dioxin from 1942 to 1984 involving a total of 5172 men at 12 different plants, it was suggested that high dioxin levels in humans are linked to cancer such as soft tissue sarcomas(Fingerhut et al. 1991). Furthermore, from a mortality follow-up of 1583 workers employed in a chemical plant in Germany that produced herbicides, including processes contaminated with TCDD, the observed increased risk of cancer mortality is associated with exposure to TCDD and supports the hypothesis that TCDD is a human carcinogen(Manz et al. 1991).

Although Hattemer-Frey and Travis(1989) reported that the total PCDD/F daily intake associated with exposure to municipal waste incinerator was about 0.3 pg/day, as a summation of the intake by inhalation, ingestion and dermal absorption exposures, this value was insignificantly small compared to the above measured or calculated daily food intakes. From our comparative results, it was found that smoking contributes to the human PCDD exposure since the intake from smoking 20 cigarettes/day is about 0.08 pg/kg/day, and its incremental cancer risk ratio accounts for approximately 6 % versus daily

food ingestion as a source.

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