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Cadmium exposure of women in general populations in Japan during 1991–1997 compared with 1977–1981

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Abstract ***Objectives:*** The Japanese people are known to have high environmental exposure to cadmium (Cd). The present survey was initiated to elucidate possible changes in the intensity of Cd exposure to the population by comparison of the present exposure level with the situation some 15 years ago. ***Methods:*** During 1991–1997, 24-h food-duplicate samples, peripheral blood specimens and morning spot urine samples were collected from 588 non smoking women from 27 survey sites in six regions, where food-duplicate and blood samples had also been obtained during 1977–1981 from 399 women. The samples were wet-ashed (after homogenization in the case of food-duplicates), and Cd in the wet-ashed samples was analyzed by inductively-coupled plasma mass spectrometry for Cd intake via foods (Cd-F), Cd concentration in blood (Cd-B) and Cd concentration in urine (Cd-U). The Cd-F and Cd-B were compared with the Cd-F and Cd-B obtained at the same sites in the 1977–1981 survey. ***Results:*** The exposure levels during 1991–1997 were such that Cd-F, Cd-B and Cd-Ucr (Cd–U after correction for creatinine concentration) were 25.5 μg/day, 1.90 μg/l and 4.39 μg/g creatinine. Comparison with the 1977–1981 survey results (i.e., 37.5 μg/day for Cd-F and 3.47 μg/l for Cd-B) showed that there were significant reductions (by 32 and 45%) in both parameters respectively during the last 15 years. The dietary route was an almost exclusive (i.e., 99% of the sum of dietary and respiratory uptake) route of Cd uptake, of which Cd in rice (11.7 μg/day) contributed about 40% of the total dietary intake. When compared among survey sites, inter-site variation in dietary Cd intake was primarily due to differences in the intake through boiled rice. Despite the recent reduction in Cd exposure, the current exposure level for Japanese people is still higher than the levels among other rice-dependent populations in Asia as well as in other parts of the world. Comparison was made between the present findings in general populations and observations among known Cd-pollution cases in Japan. ***Conclusions:*** Dietary uptake is an almost exclusive route of Cd exposure in the general Japanese population. Boiled rice is a strong determinant of variation in dietary Cd intake. Whereas there was a substantial reduction in Cd exposure among Japanese populations in the last 15 years, the current level is still high when compared internationally.

Key words Biological exposure monitoring · Blood · Cadmium environmental exposure · Food · Japanese women · Urine

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Introduction

Whereas cadmium (Cd) is ubiquitous in the environment (Nriagu 1989), human activities such as mining, industrial and, in some cases, even agricultural activities may cause pollution of the environment with this element and thus induce exposure to local human populations (Jones et al. 1987; International Programme on Chemical Safety 1992a, b). It is known that the major route of exposure to the general population is almost exclusively through foods including drinks (Vahter and Slorach 1990; Becker and Kumpulainen 1991; Barbera et al. 1993; Ikeda 1992; Lopez-Artiguez et al. 1993; Staessen et al. 1994; Zhang et al. 1997a), and in the case of

Japanese population, the staple food of the people, rice, plays a significant role as the non-occupational source of Cd exposure (Ikeda 1992). In fact, an international survey on the Cd content of rice for local consumption showed that Cd levels in rice harvested in Japan were among the highest in the world during 1977–1981 (Watanabe et al. 1989). It was also the case during 1991–1997 (Watanabe et al. 1996a) with essentially no change in the Cd content of the rice.

Our group has been conducting a nationwide survey on dietary Cd intake in the Japanese general population as well as Cd levels in blood, from 1977 up to the present time. The trend during this period is described in this report. Information on Cd levels during the years around 1980 has been reported previously (Watanabe et al. 1983, 1985). A preliminary report on time-dependent changes in Cd exposure in Japan has also been published (Watanabe et al. 1996b).

Materials and methods

Survey sites and populations

As previously described (Watanabe et al. 1983, 1985), a series of surveys was conducted in Japan during the winter seasons of 1977–1981 at 27 sites throughout the country from the most northerly Hokkaido Island to the most southerly Okinawa Islands. The second series of surveys was carried out during 1991–1997 at the same sites that the 1977–1981 survey was made (for locations, see Fig. 1). The same protocol as previously detailed (Watanabe et al. 1983, 1985; Shimbo et al. 1996a, b; Yamada et al. 1996) was followed so that possible time-dependent changes during the last 15 years could be detected by comparison. In both series, days of social or personal importance were excluded, to avoid biases due to special celebratory activities.

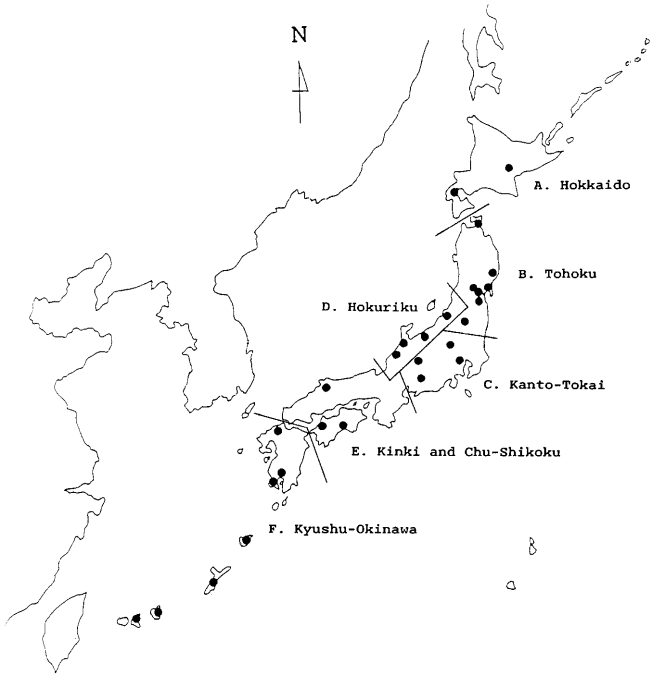

Fig. 1 Locations of survey sites, as grouped by administrative region. Regions are as shown in Table 1

Table 1 Survey sites as classified by administrative region and number of subjects in the 1977–1981 and 1991–1997 surveys

Region	Number of sites[a]	Number of subjects	
		1977–1981[b]	1991–1997[b]
A. Hokkaido	2	33	51
B. Tohoku	7	82	131
C. Kanto-Tokai	4	61	102
D. Hokuriku	4	60	99
E. Kinki and Chu-Shikoku	3	51	61
F. Kyushu-Okinawa	7	112	144
Total	27	399	588

[a] Number of sites in the region (the same for 1977–1981 and 1991–1997 surveys)
[b] 1977–1981: number of subjects in the 1977–1981 survey; 1991–1997: number of subjects in the 1991–1997 survey

Non-pregnant and non-lactating women in each survey site were invited to participate in the study as survey subjects, and non-smoking and non-habitually drinking women with no experience of occupational exposure to any heavy metals including Cd were selected. Those currently with any known clinical diseases were excluded. In practice, 399 and 588 women met the screening criteria in the 1977–1981 and 1991–1997 surveys, respectively. The numbers of women are summarized by region and by survey time in Table 1.

Sample collection and instrumental analysis for Cd

Each subject was asked to offer duplicates of her daily food-intake (Acheson et al. 1980), to allow peripheral blood samples to be taken, to agree to the collection of morning spot urine samples and to bring raw or boiled rice from her kitchen. The subject was carefully instructed to cook ordinary dishes and collect her food-duplicates quantitatively (Shimbo et al. 1996a, b; Yamada et al. 1996). The procedures used to calculate energy intake from the food-duplicates was as previously described (Shimbo et al. 1996a, b; Yamada et al. 1996). A blood sample was taken the next morning (between 9:00 A.M. and 12 noon, and without the subject having fasted) and a morning spot urine sample was also collected.

The food-duplicate (after homogenization), blood, urine and rice samples were wet-ashed by heating in the presence of mineral acids (Watanabe et al. 1982), and the wet-ash samples were subjected to metal analysis. The Cd intake via foods (including drinks) and from rice-based foods in particular (almost exclusively as boiled rice) was calculated from the Cd in the wet-ash of each homogenate or rice sample, the weight of the homogenate and the amount of rice consumed. In cases where boiled rice samples were not analyzed, Cd in boiled rice was estimated from the Cd in raw rice, based on the fact that 1 g of raw rice gives 2.4 g of boiled rice (Resources Council 1982). About a half of the material collected during 1991–1994 was the material reported in a previous publication (Watanabe et al. 1996a) and analyzed by the inductively-coupled plasma mass spectrometric method (or the ICP-MS method; Zhang et al. 1997a), this time for uniformity.

The quality of the analysis was certified by the 20th Round Robins; the detection limits were 0.1 ng/g (about 0.2 μg/day) for daily Cd intake via foods (Cd-F), 0.2 μg/l for Cd-B, and 0.02 μg/l for Cd-U. In this connection, it should be noted that the results by ICP-MS and the graphite furnace atomic absorption spectrometry (GFAAS) for lead (Pb) and Cd agree well with each other when wet-ashed samples were applied (Zhang et al. 1997a). Therefore, the 1977–1981 survey results obtained by GFAAS analysis can be compared with the 1991–1997 ICP-MS analysis data without any correction. The advantage of ICP-MS in urinalysis for metals has recently been discussed (Schramel et al. 1997).

In total, 399 pairs of samples of Cd in blood (or Cd-B) and daily Cd intake via foods (Cd-F) were available from the women in the 27 survey sites in the 1977–1981 survey, and 588 pairs in the 1991–1997 survey at the same sites (Table 1). In addition, Cd concentrations in spot urine (Cd-U) samples were available in the 1991–1997 survey. They were expressed as observed (Cd-Uob), and also after correction for urine density in terms of creatinine concentration (Cd-Ucr) (Jackson 1966) or a specific gravity of 1.016 (Cd-Usg) (Rainsford and Lloyd Davies 1965). Creatinine concentration and the specific gravity of urine were determined by colorimetry and refractometry, respectively.

Statistical analysis

The distribution of measurements were evaluated either as classified by survey site, or further combined by administrative region (Table 1). A log-normal distribution of Cd-F, Cd-B and Cd-U was previously confirmed (Watanabe et al. 1983, 1985; Abe et al. 1986; Ikeda et al. 1989; Moon et al. 1997), so that a geometric mean (GM) and a geometric standard deviation (GSD) were taken to represent the distribution. In calculating the logarithms, the measurements below the detection limits were considered as if they were half the detection limits. One-way of analysis of variance (ANOVA) was employed to detect any difference in distribution. Regression analysis was also employed.

Results

Cd-F and Cd-B in the 1991–1997 survey compared with the levels in the 1977–1981 survey

Cd-F and Cd-B in the 1991–1997 survey are summarized in Table 2 by survey region in comparison with the Cd-F and Cd-B observed at the same sites in the regions in the 1977–1981 survey. Comparison by ANOVA of Cd-F by survey site (data not shown) showed that the Cd-F varied significantly ($P < 0.01$) depending on the site, from 14.2 μg/day to 70.5 μg/day, with a highest/lowest ratio of about 5. Similarly, Cd-B by survey site also distributed in a wide range from 0.71 μg/l to 3.98 μg/l, the highest/lowest ratio being 5.6. When compared with the corresponding values in the 1977–1981 survey, the Cd-F and Cd-B in the 1991–1997 survey were significantly ($P < 0.01$ or 0.05 depending on the case) lower than the corresponding values in the 1977–1981 survey in a number of survey sites (14 sites and 22 sites in the case of Cd-F and Cd-B, respectively). It should be noted that no value in the 1991–1997 survey was significantly larger than the corresponding value in the 1977–1981 survey. Thus, the reductions in Cd-F and Cd-B were also significant ($P < 0.01$ in most cases, except for Cd-B in Hokuriku for which P was < 0.05) when compared on a regional basis (Table 2).

Correlation between 1991–1997 and 1977–1981 values

Possible correlation was examined between the 1991–1997 values and the 1977–1981 values (Table 3). The linear regression analysis after logarithmic conversion of 27 pairs of survey site measurements showed that there was a significant ($P < 0.01$) correlation in Cd-F between 1991–1997 and 1977–1981, and that the same was also the case for Cd-B during 1991–1997 and 1977–1981. The correlation was even closer with correlation coefficients of as large as 0.9 when analyzed on a regional basis. The high correlation suggests that the site or region with high Cd-F or Cd-B in the 1991–1997 survey tended also to be high in the 1977–1981 study (and

Table 3 Correlation in cadmium levels in food duplicate and in blood the 1977–1981 survey and the 1991–1997 survey

Measure	Comparison by:	No. of cases	Regression parameters[a]		
			α	β	*r*
Log10[Cd-F (μg/day)]	Site	27	0.32	0.683	0.691
	Region	6	−0.28	1.061	0.908
Log10[Cd-B (μg/l)]	Site	27	−0.16	0.812	0.487
	Region	6	−1.36	3.015	0.877

[a] α and β are regression line parameters so that $Y = (\alpha + \beta X)$, where X and Y are logarithm of the values in the 1977–1981 survey and the 1991–1997 survey, respectively; r is the correlation coefficient (all being statistically significant with $P < 0.01$)

Table 2 Cadmium levels in food duplicate and in blood in 1977–1981 and 1991–1997 surveys[a]

Region[b]	Cd-F (μg/day)		Cd-B (μg/l)	
	1977–1981	1991–1997	1977–1981	1991–1997
A. Hokkaido	33.0 (1.59)	18.7 (1.88)**	3.74 (1.35)	2.17 (1.52)**
B. Tohoku	32.4 (2.05)	23.7 (2.40)**	3.65 (1.38)	1.80 (1.91)**
C. Kanto-Tokai	29.7 (1.75)	22.2 (2.20)**	3.57 (1.44)	1.77 (1.50)**
D. Hokuriku	65.1 (1.57)	51.3 (1.69)**	3.93 (1.54)	3.41 (1.63)*
E. Kinki and Chu-Shikoku	48.9 (1.75)	26.8 (1.93)**	3.50 (1.53)	2.01 (1.59)**
F. Kyushu-Okinawa	32.5 (2.05)	20.5 (1.72)**	2.99 (1.49)	1.29 (1.81)**
Total	37.5 (1.97)	25.5 (2.14)**	3.47 (1.48)	1.90 (1.86)**
ANOVA				
Among regions	< 0.01	< 0.01	< 0.01	< 0.01
Among survey sites	< 0.01	< 0.01	< 0.01	< 0.01

*,**, indicate a significant difference ($P < 0.05$ and 0.01, respectively) between the 1977–1981 survey and the 1991–1997 survey

[a] Values are GM(GSD); the unit for GM is shown in the table (for number of cases, see Table 1)

[b] For location, see Fig. 1

Table 4 Cadmium levels in urine as studied in the 1991–1997 survey. Values are GM(GSD); the unit for GM is shown in the table

Region[a]	Cd-Uob[b] (μg/l)	Cd-Ucr[c] (μg/g cr)	Cd-Usg[d] (μg/l)
A. Hokkaido	3.07 (1. 52)	5.69 (1.39)	2.74 (1.47)
B. Tohoku	2.20 (0.24)	3.55 (2.00)	2.18 (1.98)
C. Kanto-Tokai	1.93 (1.92)	3.53 (1.70)	2.18 (1.62)
D. Hokuriku	3.83 (2.18)	7.03 (2.10)	4.07 (2.08)
E. Kinki and Chu-Shikoku	2.62 (2.18)	5.29 (1.94)	2.84 (1.98)
F. Kyushu-Okinawa	2.29 (2.09)	3.79 (1.77)	2.35 (1.81)
Total	2.50 (2.14)	4.39 (1.96)	2.59 (1.92)
ANOVA			
Among regions	< 0.01	< 0.01	< 0.01
Among survey sites	< 0.01	< 0.01	< 0.01

[a] For location, see Fig. 1
[b] Cd-U as observed
[c] Cd-U as corrected for creatinine concentration
[d] Cd-U as corrected for a specific gravity of 1.016

therefore most possibly in the past in general), and that the reverse should also be the case.

Cd-U levels in the 1991–1997 survey

Cd-U concentrations were determined in the 1991–1997 survey and the results are summarized in Table 4 as Cd-Uob, Cd-Ucr or Cd-Usg. The grand GM for a total of 588 urine samples was 2.50 μg/l for Cd-Uob, 4.39 μg/g creatinine for Cd-Ucr and 2.59 μg/l for Cd-Usg. Variation in Cd-U on a site basis was significant ($P < 0.01$) regardless of the correction for urine density when examined by ANOVA. No Cd-U data were available in the 1977–1981 survey, and unfortunately, therefore, possible time-dependent changes in Cd-U could not be examined.

Role of rice as the dietary Cd source

Food-duplicate samples from the 588 women on average contained 1,848 kcal of energy, the consumption of boiled rice was 439 g/day and the energy from the boiled rice was 650 kcal/day (Table 5). Analysis for Cd in food-duplicates gave 25.5 μg/day for Cd-F. The measurement of Cd content in boiled rice multiplied by the amount of boiled rice consumed by individuals showed that the GM of Cd taken as a contaminant in boiled rice (or Cd-BR) was 10.8 μg/day (Calculation A in Table 5), or 42.4% of Cd-F.

Cd intake via all foods (Cd-F) was classified into two groups of Cd intake via boiled rice (Cd-BR) and Cd intake via foods other than boiled rice (i.e., Cd-NBR). Regression analysis on a survey site basis ($n = 27$) of Cd-BR with Cd-F (both after logarithmic conversion) showed that Cd-BR correlated significantly ($r = 0.868$, $P < 0.01$) with Cd-F, whereas Cd-NBR did not show any significant correlation ($P > 0.10$) with Cd-F. The slope of the regression line with Cd-NBR was less than one third of that with Cd-BR (Fig. 2). In addition, multiple regression analysis (after logarithmic conversion) on an individual basis (i.e., $n = 588$) taking Cd-F as a dependent variable, and Cd-BR and Cd-NBR as two independent variables disclosed that only Cd-BR was influential, with a partial correlation coefficient of 0.667 ($R^2 = 0.445$). Similar analyses on a survey site ($n = 27$) or regional basis ($n = 6$) gave the same results with even greater R^2s of 0.655 and 0.805, respectively. These findings all together indicate a major role of rice as the dietary source of Cd for the study populations.

Table 5 Rice as an energy source as well as the source of dietary cadmium intake

Item	(Unit)	1977–1981	1991–1997
No. of subjects		399	588
Energy intake	(cal)	2173 ± 504	1848 ± 428**
Boiled rice intake	(g/day)	635 ± 223	439 ± 157**
Energy intake from rice	(cal)	941 ± 330	650 ± 232**
Energy intake from rice	(%)	43.3%	35.2%
Cadmium intake from foods	(μg/day)	37.5(l.97)	25.5(2.14)**
Cadmium from rice	(μg/day)		
Calculation A[a]			10.8(3.53)
Calculation B[b]		13.9	10.2
Cadmium intake from rice	(%)		
Calculation A			42.4%
Calculation B		37.0%	40.0%

[a] Based on the measurement of Cd in boiled rice samples collected in the present study
[b] Geometric mean Cd concentrations in raw rice collected in Japan at the beginning of the 1980s and in the middle of the 1990s; 52.5 ng/g (Watanabe et al. 1989) and 55.7 ng/g (Watanabe et al. 1996), respectively, were employed, together with an estimation that 100 g raw rice gives 240 g boiled rice (Resources Council 1982)

Discussion

Typical Cd exposure indicator values reported in literature for four known Cd-polluted areas in Japan are summarized in Table 6. It was estimated that the dietary Cd exposure was as high as 604 μg/day in the Jindzu River basin (Environment Agency 1972; Kitamura 1972) where an endemic of Itai-itai disease was observed. Cd-Ucr in urine of the patients, measured in 1967 and 1975, were also high, around 30 μg/g creatinine (Nogawa and Kido 1993). In another polluted area, Tsushima Island, Cd-F was estimated to be nearly 500 μg/day

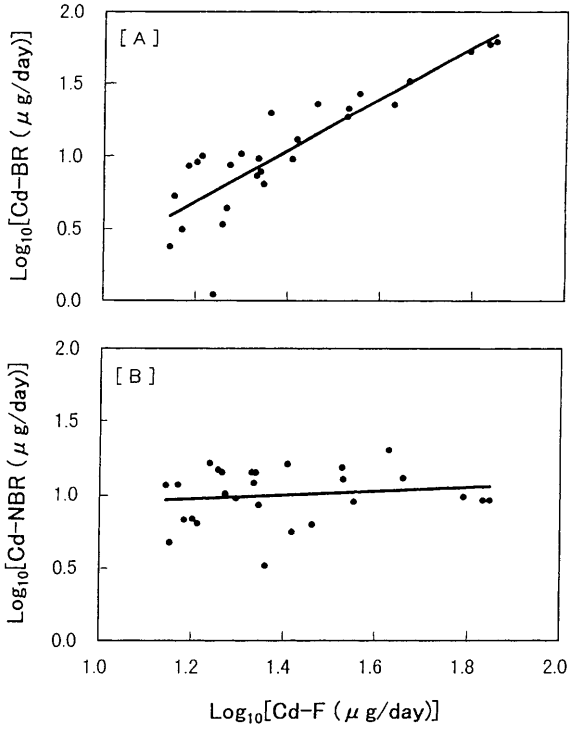

Fig. 2 Contribution of boiled rice and other foods as the Cd source in total dietary Cd intake. The amount of Cd ingested as **A** boiled rice (shown as Cd-BR) and **B** foods other than boiled rice (Cd-NBR) are compared with total dietary Cd intake in the 1991–1997 survey. The line in the middle is a calculated regression line, $Y = -1.44 + 1.78X$ ($r = 0.868$, $P < 0.01$, $n = 27$) in **A** and $Z = 0.81 + 0.13X$ ($r = 0.147$, $P > 0.10$, $n = 27$) in **B**, where X, Y and Z are logarithm of geometric means for each site of dietary Cd intake (Cd-F), Cd intake via boiled rice (Cd-BR) and Cd intake via foods other than boiled rice (Cd-NBR), respectively

(Environment Agency 1972; Kitamura 1972), 150 μg/day (Tsuchiya and Iwao 1978), or about 200 μg/day in the past which was reduced to 100 μg/day or less after the replacement of contaminated soil with clean soil (Iwata et al. 1993). The highest Cd-Ucr reported was 11.2 μg/g creatinine for women (Tohyama et al. 1986).

A similar Cd-Ucr level of about 10 μg/g creatinine was reported for the residents in the third polluted area, the Kakehashi River basin (Kido et al. 1988). In Kosaka, the fourth polluted area, Cd-F was estimated to be 90.5 μg/day by Tsuchiya and Iwao (1978). Compared with these values reported for the residents of polluted areas in Japan, the levels observed in the present study, i.e., 25.5 μg/day as Cd-F (Table 2) and 4.39 μg/g creatinine as Cd-Ucr (Table 4) are about one fourth and one half of the lowest values reported for contaminated areas, respectively (Table 6).

Theoretically, both the lungs and the dietary tract are routes of Cd intake (Table 7).The Environment Agency (1981, 1997) has been reporting Cd concentrations in the general atmosphere Japan, e.g., 2–10 and 0.6–2.9 ng/m^3 for 1981 and 1997, respectively. With the two assumptions that the respiratory volume is 15 m^3/day and that the uptake ratio for Cd in the lungs is about 50% (Ikeda 1992), it is possible to estimate the daily Cd uptake in the lungs as 2–10 (or 0.6–2.9) ng/m^3 × 15 m^3/day × 50/100 = 0.03 (or 0.01) μg/day for 1991–1997 (values for 1977–1981 in parenthesis). Similarly, dietary uptake will be 2.85 (or 1.91) μg/day for 1991–1997 (1977–1981 in parenthesis) when an absorption ratio is assumed to be 7.5% (Ikeda 1992). When the Cd uptake through inhalation and ingestion are compared, it is apparent that the uptake via the dietary tract is almost exclusive (about 99%) for Cd uptake throughout the two surveys.

Table 6 Typical values reported to show the extent of Cd exposure in known Cd-polluted areas in Japan[a]

Location	Cadmium in		Reference
	Food[b]	Urine[c]	
Jinzdu River basin (Toyama Prefecture)	604		Environment Agency 1972; Kitamura 1972
		1967 31[d]	Nogawa and Kido 1993
		1975 27[d]	
Tsushima island (Nagasaki Prefecture)	493		Environment Agency 1972; Kitamura 1972
	148.7		Tsuchiya and Iwao 1978
	Kashine, 1969 213		Iwata et al. 1993
	Kashine, 1983 106		Iwata et al. 1993
	Total 1983 77		Iwata et al. 1993
		Men 6.6	Tohyama et al. 1986
		Women 11.2	Tohyama et al. 1986
		1982 8.5	Iwata et al. 1993
		1986 6.0	Iwata et al. 1993
Kakehashi River basin (Ishikawa Prefecture)	97.2		Tsuchiya and Iwao 1978
		1981 10.0	Kido et al. 1988
		9.6	Kido et al. 1988
Kosaka (Akita Prefecture)	90.5		Tsuchiya and Iwao 1978
		8.0	Kojima 1975

[a] Values in the table are assumedly AM
[b] Cd-F in μg/day
[c] Cd-U in μg/g creatinine
[d] Itai-itai disease patients

Table 7 Cadmium exposure by route of entry among non-smoking adult Japanese women with no occupational Cd exposure

Survey time	Route of entry	Source	Conc. in air (ng/m^3)[a]	Intake (μg/day)	Uptake[b] (μg/day)	Total[c] (μg/day)
1980	Ingestion	Food	–	38.0	2.85	2.88
	Inhalation	Air	2–10	0.067	0.03	
1990	Ingestion	Food	–	25.5	1.91	1.92
	Inhalation	Air	0.6–2.9	0.020	0.01	

[a] The minimum and the maximum concentrations, of which GM was taken as the representative value for calculation; cited from Environment Agency, Japan (1981, 1997)
[b] An absorption rate of 7.5% in the digestive tract was assumed for the ingestion, and a respiration volume of 15 m^3/day and an absorption rate in the lungs of 50% were assumed for the inhalation (Ikeda 1992)
[c] Combination of exposures via ingestion and via inhalation

It was also known that rice was the major source of Cd exposure in Cd-polluted areas in Japan so that Cd in rice was often taken as an indicator of dietary Cd intake (e.g., Kido and Nogawa 1993; Hochi et al. 1995). Rice was a substantial Cd source also in non-polluted areas (Ikeda 1992). The present analysis showed that Cd from Cd-BR contributed 46% of the total dietary Cd intake (Table 5). Because no boiled rice samples were available in the 1977–1981 survey, an attempt was made to estimate Cd-BR from the Cd contents in raw rice.

Rice samples collected in Japan at the beginning of 1980s and then in the middle of 1990s contained 52.5 (Watanabe et al. 1989) and 55.7 ng/g raw rice (Watanabe et al. 1996a), respectively, as the GM. Taking advantage of these concentrations in combination with the amounts of boiled rice consumed in the 1977–1981 and 1991–1997 surveys, together with the fact that 100 g of raw and boiled rice contain 356 and 148 kcal respectively (Resources Council 1982) and that therefore 100 g of raw rice will give 240 g boiled rice, it was possible to estimate that consumption of 439 g boiled rice/day (1991–1997) or 635 g/day (1977–1981) containing 55.7 (52.5) ng Cd/g of raw rice will result in an intake of 10.2 μg/day for 1991–1997 or 13.9 μg Cd/day for 1977–1981 (Calculation B in Table 5) via boiled rice. Thus, a substantial reduction was noted in the last 15 years in Cd-F as well as Cd intake from rice; the reduction was associated with reductions in total energy intake, boiled rice consumption and energy intake from boiled rice (Table 5).

Further comparison of Cd-BR with Cd-F shows that the percentage Cd intake via boiled rice [(Cd-BR/Cd-F)] was 40.0% in 1991–1997, whereas it was 37.0% in 1977–1981, indicating that Cd-BR accounted for almost 40% of Cd-F throughout the time period studied (Table 5). It was estimated that the contribution of rice as the dietary Cd source was as high as 73.3% in the Jindzu River basin (Environment Agency 1972; Kitamura 1972). It was 73.9% in the Kakehashi River basin (Tsuchiya and Iwao 1978), 52.3% in Kosaka (Tsuchiya and Iwao 1978), 36.3% (Tsuchiya and Iwao 1978) and 31.4% in Tsushima Island (Environment Agency 1972; Kitamura 1972). The percentages for the two polluted areas of the Jinzdu and Kakehashi River basins were almost twice as high as that in the present observation. A relatively low percentage for Tsushima people was due to their dietary dependence not only on rice but on potatoes (assumedly sweet potatoes) for their daily energy requirements (Environment Agency 1972; Kitamura 1972).

A recent report from the Jundzu River area (Fan et al. 1998) showed that the replacement of Cd-polluted rice paddy soil with a clean one brought down the Cd level in rice (harvested from the paddy) from the original levels of 240–440 ng/g (as the GM) to 40–150 ng/g, or 20% of the original on average. Taking this reduction rate of 20% in combination with the reduced rice consumption (from 635 g boiled rice/day to 439 g/day; Table 5), it is possible to estimate that rice-based Cd intake after the soil replacement will be 61 μg/day ($= 604$ μg/day (Table 6) $\times 73.3/100 \times 439/635 \times 20/100$). Assuming that there was no change in Cd intake from foods other than rice, the role of rice as a dietary Cd source would be about 30% of the total Cd intake. Similar calculation in the Kakehashi River basin and Kosaka gave 28% and 13%, respectively, indicating a shift of the major Cd source from rice to other foods.

Table 8 Dietary exposure to cadmium, and cadmium concentration in blood and urine among nonsmoking adult women in the cities in Asia[a]

Location	Cadmium in:			Reference[e]
	Food[b]	Blood[c]	Urine[d]	
Japan[f]				
1977–1981	37.5	3.47	–	The present study
1991–1997	25.5	1.90	4.39	The present study
Bangkok	7.1	0.41	1.40	Zhang et al. 1999
China[g]	9.9	1.07	2.30	Zhang et al. 1997[b]
Korea[h]	21.2	1.39	2.26	Moon et al. 1998
Kuala Lumpur	9.0	0.74	1.51	Moon et al. 1996
Manila	14.2	0.47	1.21	Zhang et al. 1998
Tainan	9.7	0.83	1.59	Ikeda et al. 1996

[a] Values in the table are GM. Values for Kuala Lumpur, Manila and Tainan were recalculated, taking only those for which the data on all three items are available
[b] Unit (μg/day)
[c] Unit (μg/l)
[d] Unit (μg/g creatinine)
[e] Except for cadmium in urine, which was analyzed for the present study utilizing urine samples collected at the time of studies cited and kept frozen thereafter
[f] A group of 27 sites
[g] A group of four cities in Mainland China
[h] A group of four sites

Table 9 Dietary exposure to cadmium, and cadmium concentration in blood and urine among general populations in various areas in the world[a]

Location	Cadmium in:			Reference
	Food[b]	Blood[b]	Urine[b]	
Belgium	14.6[c]			Buchet et al. 1983
		M[d]:0.96[e]	0.5 g[e]	Staessen et al. 1991
		W[d]:0.93[e]	0.49[e]	Staessen et al. 1991
		M:1.03	0.90[f]	Sartor et al. 1992
		W:1.14	0.80[f]	Sartor et al. 1992
		0.82	0.69	Roels et al. 1993
		0.39	0.66	Staessen et al. 1994
China[g]	W:7.0[c]	W:0.54[c]		Vahter and Slorach 1990
		M:0.94		Qu et al. 1993
		W: 0.83		Qu et al. 1993
			0.40[f]	Nordberg et al. 1997
Croatia	W:7.8[c]	W:0.66[c]		Vahter and Slorach 1990
Europe[h]			0.59	Taylor et al. 1997
Germany	M:7.4[c]			Müller and Anke 1994
	W:6.9[c]			Müller and Anke 1994
		1.0	0.86	Jung et al. 1993
Italy		0.36		Alessio et al. 1993
Japan	W:19.8[c]	W:1.49[c]		Vahter and Slorach 1990
			W:5.52	Kido et al. 1995
			W:3.60	Hayano et al. 1996
			W:1.3	Yamanaka et al. 1998
Singapore	0.26			Chia et al. 1994
Spain	18			Lopez–Artiguez et al. 1993
	11			Barbera et al. 1993
Sweden	W:8.3[c]	W:0.26[c]		Vahter and Slorach 1990
	12			Becker and Kumulainen 1991
		0.45		Lagerkvist et al. 1993
The Netherlands	0.68	0.50–0.87		van Sittert et al. 1993
USA	M+W:0.41			Hovinga et al. 1993

[a] Both GM and AM (sexes were not identified in some cases) are cited as reported
[b] Unit (μg/day for Cd-F, μg/l for Cd-B and μg/g creatinine for Cd-U)
[c] Geometric mean, estimated from the arithmetic mean and the arithmetic standard deviation by the moment method (Sugita and Tsuchiya 1995)
[d] M and W stand for men and women, respectively
[e] Including smokers
[f] Unit (μg/l)
[g] Mainland
[h] Integrated data from Germany and Belgium

Table 8 compares Cd-F, Cd-B and Cd-Ucr in the present study with the values for other Asian cities (Ikeda et al. 1996; Moon et al. 1996, 1998; Zhang et al. 1997b, 1998, 1999), where people also depend heavily on rice for the energy of daily life. It should be noted that these studies, including the present one, were carried out under the same protocol and that the analysis was done in a single laboratory under the same analytical conditions (Zhang et al. 1997a). It is clear that the Cd exposure in 1991 in Japan was the highest of all measurements (i.e., Cd-F, Cd-B and Cd-U) in the survey locations, although there had been substantial reduction in the exposure since 1977–1981 (Tables 2, 4). The Cd exposure levels are generally low in other parts of Asia with the one possible exception of Korea where Cd-F and Cd-B were the second highest and Cd-Ucr the third (Moon et al. 1998).

Recent reports from other groups are reviewed in Table 9. It should be noted that the studied population varied in sex and age (although they were adult) and that analytical methods (including the methods for food sample collection) were also different, depending on the reports. Nevertheless, three reports from Japan (Kido et al. 1995; Hayano et al. 1996; Yamanaka et al. 1998) agree with the present observation in the sense that Cd-Ucr values are substantially higher than the levels reported for other areas. The paper from Singapore (Chia et al. 1994) reported a low Cd-Ucr there in agreement with low levels for parts of east Asia other than Japan (Table 5), whereas Cd-Ucr (0.48 μg/l) reported by Nordberg et al. (1997) for Mainland China was lower than the present value (2.30 μg/g creatinine; Table 9) observed in major cities on the continent, even when the difference in the unit is taken into account.

Nevertheless, it seems prudent to summarize that the current level of environmental exposure to Cd in Japan is still higher than in other areas of the world, despite the substantial reduction over the recent years. Possible roles of volcano activities (Bargagli et al. 1991) in prehistoric times as well as the application of fertilizers in the recent past (Hutton and Symon 1986; Jones et al. 1987) have previously been discussed as the sources of not local but general soil contamination (Ikeda et al. 1994).

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