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Urinary cadmium and *N*-acetyl-β-D-glucosaminidase excretion of inhabitants living in a cadmium-polluted area

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Summary. Urinary cadmium (Cd), *N*-acetyl-β-D-glucosaminidase (NAG), and creatinine levels were determined for 400 inhabitants living in Cd-polluted districts of Annaka City in Gunma Prefecture, Japan. The Cd pollution was mainly due to falling dust from a zinc smelter factory according to seasonal winds. The Cd-polluted areas were divided into five administrative districts around the factory. The geometric mean of the urinary Cd level of the inhabitants in the five districts, Nodono, Iwai, Ooya, Nakajyuku, and Itahana, were 2.95, 2.66, 2.45, 1.97, and 1.67 μg/g creatinine, respectively. The geometric means for Nodono and Iwai are statistically larger than that for Itahana ($P < 0.01$). These results were well explained by the wind direction and proximity to the factory. In addition, a two-way analysis of variance on the urinary Cd level was made using place of residence and smoking habits. The effect was mainly due to the place of residence, and no interactions were found. There were statistically significant differences in NAG excretion among the five groups, but no difference could be found between two groups using a multiple comparison. Pearson's correlation coefficient between the logarithm of urinary Cd content and that of NAG was 0.203, but statistically significant ($r = 0.462$ without creatinine correction; $P < 0.01$). The present results indicate the association of urinary Cd and NAG levels in a Cd-exposed population whose geometric mean of urinary Cd content is about 2 μg/g creatinine. We recommend a continuous survey of the minimum health effects of the Cd pollution using urinary levels of Cd and protein including NAG.

Key words: Cadmium-polluted area – Field survey – *N*-acetyl-β-D-glucosaminidase – Geographic location – Health examination

Introduction

The health effect of cadmium (Cd) in and outside industry has been investigated in many countries, and several biological indicators have been used to estimate its body burden and health effects [2, 5–7, 9, 11, 16, 21, 29, 31]. The kidney cortex is a critical organ affected by long-term Cd exposure, and the accumulation of Cd results mainly from eating grains and vegetables. The body burden of Cd is partly determined by smoking, age, and residence [16]. However, smoking seems to have a smaller effect on the urinary Cd increase than on the blood Cd level [5], which reflects recent exposure.

In the past, the people living in Cd-polluted areas in Japan were surveyed exclusively where severe health effects had been reported, such as "Itai-Itai disease" in the Jinzu River basin. Rice is a staple food for the Japanese. The average Cd intake of the Japanese living in nonpolluted areas is more than twice as high as that in European, American, and Asian people [28]. These facts indicate the importance of a health survey in other low-level pollution areas of Cd in Japan.

A zinc smelter factory was set up in Annaka City (pop. 47000; Fig. 1) in Gunma Prefecture in 1937. Four districts in Annaka City (Itahana, Nakajyuku, Iwai, and Nodono) were identified as Cd-polluted areas to be followed up in a study conducted by the Ministry of Health and Welfare in 1969. Health examinations were held from 1969 to 1975 for all inhabitants over 40 years of age. From 1976, other specified districts were added, and the health examination was continued every year by specifying a few more districts in the city in turn. As these health checks were not intended to follow the same population for many years, the relationship of the Cd body burden and health effects, such as kidney damage, could not be clarified.

The Cd pollution in this area is mainly due to dust from the factory falling onto the fields from 1930s to 1970s. Waste-water from the factory was not responsible for the pollution. A strong northwest wind blows in winter, and a gentle southeast one blows during the summer season. The Cd from the rice contributes 40% of the daily intake in normal Japanese [31]. In the first half of the 1970s, farmers of this area took in 280 μg of Cd on average, which was about five times higher than the average Japanese Cd intake [13, 27]. In 1988, the range of the Cd content in brown rice of 44 samples of Cd-polluted areas in Annaka City was 85–1480 ppb, and the average

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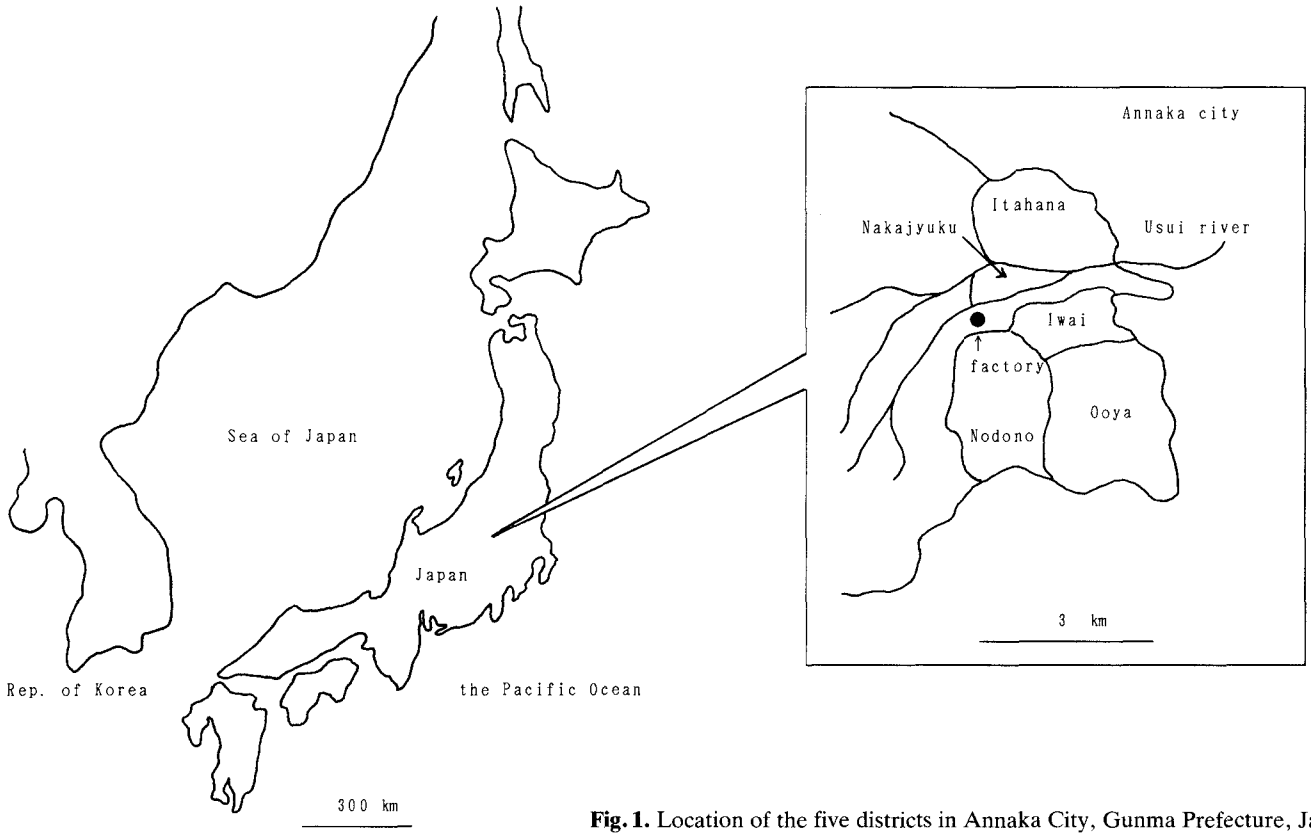

Fig. 1. Location of the five districts in Annaka City, Gunma Prefecture, Japan

was 358 ppb in contrast to 50 ppb, which was the upper limit of the control sample in a highland area of Ibaraki Prefecture [14]. In the same period, Rivai et al. measured the Cd content in 64 unpolished rice samples in the Kanto district which ranged from 2.1 to 321.5 ppb with a geometric mean of 49.0 [19]. The mean Cd level of polluted rice in the Jinzu River basin in Toyama Prefecture was 620 ppb in 1972 [14]. As a first measure, polluted rice was not allowed to be sold commercially in the markets to decrease the Cd level of crops and vegetables. For the second measure, a replacement program of the rice field soil was promoted by the government.

The critical concentration of urinary Cd was proposed to be 10 μg/g creatinine [30]. The renal damage is mostly irreversible once it occurs and is accelerated by aging [20, 22]. We have already found that a dose-effect relationship exists between urinary Cd and *N*-acetyl-β-D-glucosaminidase (NAG) at a Cd excretion of around 1 μg/g creatinine or more [9, 11]. Thus, the Cd body burden and health effect could be estimated very accurately using the two indicators previously mentioned. We tried to evaluate the level of the body burden of Cd and its renal effects by conducting a health survey of the inhabitants living around the factory.

Subjects and methods

Subjects examined were inhabitants over 40 years of age living in Annaka City. Five districts, Itahana, Nakajyuku, Iwai, Nodono, and Ooya, were targeted, which surround the smelter factory. The location of each district is shown in Fig. 1. Itahana and Nakajyuku are situated to the northeast or north of the factory, Nodono and Ooya to the south or southeast, and Iwai to the east.

Spot urine samples were collected in the morning from the subjects during a periodic health examination for adults required by law. A 1-ml sample of urine was added to the same amount of 2.5 *N* HNO_3 for the Cd measurement. The Cd content was determined by a flameless atomic absorption spectrophotometer (Hitachi Model 180-30). Specimens were stored at 4°C for a few days before the measurement. The NAG level was determined by a colorimetric assay [17]. The creatinine level was determined using the Folin-Wu method [3] within a month for the purpose of urine correction.

Results

Sex, age, and place of residence of the subjects are shown in Tables 1 and 2. The sex ratio between the five districts was the same.

The relation between urinary Cd or NAG and age was analysed (Table 1). The urinary Cd level changed with age according to the regression equation: $\log_{10}(\text{Cd}) = 0.0048 \times \text{age} - 0.053$ for men and $\log_{10}(\text{Cd}) = 0.0067 \times \text{age} - 0.055$ for women. The slope of the latter regression line was about 40% larger than that of the men. The urinary NAG level increased with age following the regression equation; $\log_{10}(\text{NAG}) = 0.0057 \times \text{age} + 0.21$ for men and $\log_{10}(\text{NAG}) = 0.0069 \times \text{age} + 0.14$ for women. The urinary NAG excretion of men is larger than that of women to 70 years of age (Table 1).

The mean urinary Cd concentration of subjects from all five districts were compared after logarithmic trans-

Table 1. Geometric mean and standard deviation of urinary cadmium and *N*-acetyl-β-D-glucosaminidase of inhabitants over 40 years of age of five districts in a cadmium-polluted area in Gunma prefecture, Japan

Age	*n*	Male ($n = 120$)				*n*	Female ($n = 280$)			
		Cd (μg/l)	NAG (unit/l)	CdI (μg/g cr)	NAGI (unit/g cr)		Cd (μg/l)	NAG (unit/l)	CdI (μg/g cr)	NAGI (unit/g cr)
40s	14	1.10, 1.95	3.46, 2.21	0.94, 1.74	2.95, 1.95	60	1.10, 2.55	2.15, 2.30	1.49, 2.02	2.94, 2.18
50s	21	2.21, 2.37	3.34, 2.36	2.20, 1.87	3.33, 1.61	53	1.71, 2.27	2.38, 2.28	2.48, 1.90	3.45, 2.00
60s	48	1.73, 2.06	3.45, 1.97	1.87, 1.92	3.74, 1.74	91	1.65, 2.16	2.22, 2.35	2.60, 1.92	3.50, 1.98
70s	32	1.36, 2.31	3.23, 2.30	1.94, 2.04	4.60, 1.58	66	1.56, 2.64,	2.83, 2.23	2.52, 2.04	4.58, 1.90
>80	5	1.34, 3.15	3.06, 2.26	1.68, 2.22	3.84, 1.58	10	1.06, 2.42	3.14, 1.64	2.40, 2.17	7.11, 1.67
	120	1.59, 2.25	3.36, 2.14	1.78, 2.00	3.77, 1.71	280	1.48, 2.42	2.40, 2.28	2.27, 2.04	3.67, 2.05

Cd, urinary cadmium; NAG, urinary *N*-acetyl-β-D-glucosaminidase; CdI, urinary cadmium adjusted by creatinine; NAGI, urinry *N*-acetyl-β-D-glucosaminidase adjusted by creatinine (cr)

Table 2. Number, age, urinary Cd and NAG levels of the adult inhabitants in five districts of a Cd polluted area in Gunma prefecture, Japan. Prevalence of NAG over the upper limit of reference value is also described.

District	*n* (M, F)	Age (years)	Cd[a] (μg/l)	CdI[a] (μg/g cr)	NAG[a] (unit/l)	NAGI[a] (unit/g cr)	NAG over 4.2 unit/l (%)	NAG over[a] 2.4 unit/g cr (%)
Nodono	80 (25, 55)	64.6, 9.20	2.01, 2.07**	2.95, 1.86**	2.33, 2.54	3.42, 2.02	32.5	75.0
Ooya	21 (9, 12)	62.7, 12.18	1.59, 2.20	2.45, 1.89	2.84, 2.73	4.38, 1.93	33.3	71.4
Nakajyuku	10 (3, 7)	64.8, 4.42	1.33, 1.87	1.97, 1.70	1.78, 2.96	2.64, 2.33	20.0	70.0
Iwai	82 (27, 55)	60.5, 11.92	1.92, 1.98**	2.66, 1.85**	2.22, 2.74	3.08, 2.39	24.4	70.7
Itahana	207 (56, 151)	62.0, 11.30	1.23, 2.55[b]	1.67, 2.05[b]	3.02, 1.85	4.10, 1.67	29.0	85.5
	400 (120, 280)	62.3, 11.03	1.51, 2.37	2.11, 2.04	2.65, 2.27	3.70, 1.95	28.8	79.2

Geometric mean and standard deviation are calculated for Cd, CdI, NAG, and NAGI. Arithmetic mean and standard deviation are calculated only for age in years

n (M, F), male and female

[a] There was statistically significant differences of Cd, CdI, NAG, NAGI, and prevalence of increased NAGI among the five districts

[b] Urinary Cd level in subjects from Nodono and Iwai was significantly larger than that of Itahana by the Ryan's multiple comparison

** $P < 0.01$

Table 3. Urinary Cd excretion by smoking habit of inhabitants over 40 years of age of five districts in a Cd-polluted area in Gunma prefecture, Japan

Group	Male ($n = 118$)				Female ($n = 273$)			
	n	Age (years)	Cd (μg/l)	CdI (μ/g cr)	*n*	Age (years)	Cd (μg/l)	CdI (μg/g cr)
No	37	63.0, 11.08	1.42, 2.29	1.56, 2.06	244	61.4, 11.27	1.48, 2.47	2.29, 2.07
Past	23	67.9, 7.83	1.33, 2.30	1.83, 1.92	5	66.6, 8.26	1.33, 2.63	2.17, 2.24
Smoke	58	63.7, 10.45	1.86, 2.16	1.96, 1.97	24	62.0, 12.39	1.53, 2.20	2.05, 1.91
	118	64.3, 10.28	1.66, 2.24	1.80, 1.99	273	61.6, 11.31	1.48, 2.44	2.27, 2.05

Geometric mean and standard deviation are calculated for Cd and CdI; arithmetic mean and standard deviation are calculated only for age in years

No, nonsmokers; Past, ex-smokers; Smoke, current smokers

formation of the data. The geometric mean value of the urinary Cd excretion was arrayed: Nodono > Iwai > Ooya > Nakajyuku > Ithahana. A multiple comparison using the Ryan method was carried out; the geometric means of urinary Cd level of Nodono and Iwai are statistically larger than that of Itahana ($P < 0.01$) (Table 2). The range of urinary Cd level in Nodono and Iwai was 0.4–14.6 μg/g creatinine, and that of Itahana was 0.2–10.2.

The mean urinary NAG activity of the five districts was compared after logarithmic transformation as for Cd and was arrayed: Ooya > Ithahana > Nodono > Iwai > Nakajyuku in that order. There were statistically significant differences of NAG excretion among the five groups, but a multiple comparison by the Ryan method could not be done (Table 2). The prevalence of NAG activity over 4.2 unit/l or 2.4 unit/g creatinine was examined. There were statistically significant differences of the pre-

Table 4. A two-way analysis of variance using living place and smoking habits on the effect of urinary cd excretion

Sex	Source	Logarithm of Cd (μg/l)		Logarithm of Cd (μ/g cr)	
		F value	*P* value	*F* value	*P* value
Both	Place: A	26.60	<0.001	52.07	<0.001
	Smoke: B	2.00	0.158 n.s.	0.859	0.354 n.s.
	A × B	1.14	0.287 n.s.	2.59	0.108 n.s.
Male	Place: A	3.33	0.071 n.s.	10.58	<0.01
	Smoke: B	2.08	0.152 n.s.	1.09	0.300 n.s.
	A × B	1.36	0.246 n.s.	1.17	0.282 n.s.
Female	Place: A	23.00	<0.001	41.63	<0.001
	Smoke: B	0.389	0.533 n.s.	0.020	0.887 n.s.
	A × B	0.080	0.778 n.s.	0.201	0.654 n.s.

Five districts were divided into two places by the position around the factory and wind direction: Itahana and other districts except Nakajyuku. Smoking habits were also divided into two categories: the current smokers and the others. Sex difference was also considered in this analysis

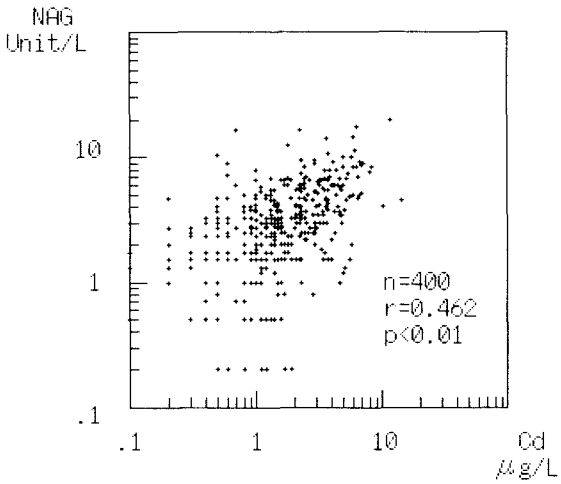

Fig. 2. Relationship between urinary levels of Cd and NAG of the inhabitants in a Cd-polluted area in Gunma Prefecture, Japan

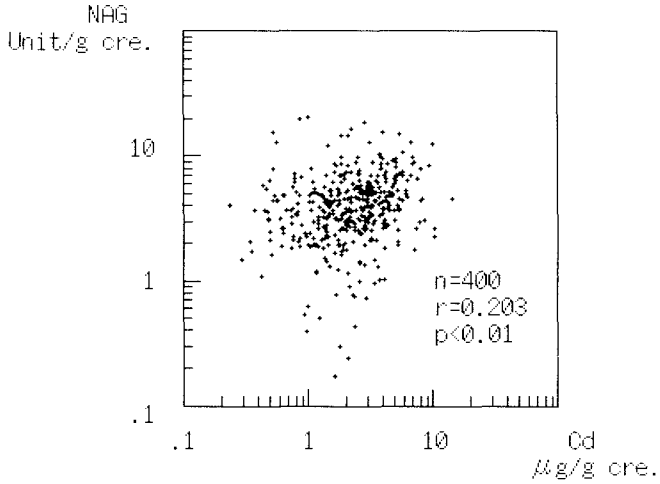

Fig. 3. Relationship between urinary levels of Cd and NAG of the inhabitants in a Cd-polluted area in Gunma Prefecture, Japan, after creatinine adjustment

valence of NAG excretion adjusted by creatinine over the reference value among the five groups, but multiple comparison using the Ryan method could not be applied (Table 2).

Effect of smoking on the urinary Cd excretion was examined. There were no differences in the mean values between nonsmoker, ex-smoker, and current smoker groups (Table 3). There was no clear dose-effect relationship between the Brinkman Index and the urinary Cd level. The former was calculated as the average number of cirgarettes smoked per day multiplied by smoking years [4].

A two-way analysis of variance on the urinary Cd excretion was done using the place of residence and smoking habits. The sample size was too small to classify by sex, district of residence, and smoking habits, so two factors (place of residence and smoking habits) were recategorized by the geographical location and smoking habits at present. The effect was mainly by place of residence, and no interactions were found (Table 4).

Table 5. Partial correlation coefficients excluding the influence of age between urinary levels of Cd and NAG in men and women. Pearson's correlation coefficients are also listed in parentheses

Sex	*n*	Logarithm of Cd (μg/l) and that of NAG (unit/l)	Logarithm of Cd (μ/g cr) and that of NAG (unit/g cr)
Male	120	0.519** (0.520)	0.206* (0.238)
Female	280	0.439** (0.443)	0.150* (0.201)

Statistical significance of partial correlation coefficients between Cd and NAG: $*P<0.05$, $**P<0.01$

Pearson's correlation coefficients between the logarithm of urinary concentration of Cd and that of NAG were small but statistically significant ($r=0.203$ with creatinine correction and 0.462 without correction, $n=400$, $P<0.01$) (Figs. 2, 3). After separating men and women, the partial correlation coefficients were analysed to exclude the influence of age. The logarithm of urinary

Cd excretion showed a statistically significant correlation with that of NAG in men ($r = 0.206$ with creatinine correction and 0.519 without, $n = 120$) and in women ($r = 0.150$ with creatinine correction and 0.439 without, $n = 280$) (Table 5).

Discussion

The sensitivity of the method of Cd measurement was never satisfactory before 1975, when an overestimation was ubiquitous, as the flameless atomic absorption spectrophotometer (FAA) had not been popular for Cd analysis [6, 7, 16]. Thus, any comparison with data of that time should be done carefully. In addition, the relationship between the Cd content in rice and that in the soil of the rice field was weak [14, 18, 23, 26].

In this survey, there was no case of Cd-induced renal dysfunction, and the correlation coefficient between urinary Cd and NAG levels was small but still significant. The subjects in the present study excrete 2.11 μg/g creatinine of mean urinary Cd, and 4 (1.0%) subjects excrete more than 10 μg of Cd/g creatinine. These four are all women aged around 70 years. The soil replacement project for rice fields finished in 1988 in this Cd-polluted area. Brown rice containing 0.4 ppm or more of Cd was not allowed to be sold and eaten, and that containing 1 ppm or more of Cd was not purchased by the government. Polluted rice containing 0.4–1 ppm of Cd was used for making glue, etc. The soil producing polluted rice was specified to be a polluted rice field, to which a soil replacement project was assigned. The replacement of the topsoil from polluted rice fields was conducted to eliminate the possibility of producing polluted rice. Iwata et al. reported on this project and found that the daily intake of Cd of the farming families was reduced from 213 to 106 μg Cd after the project [8]. Dry fields were not included in this government project, and families who eat homegrown vegetables from dry fields may have a higher daily intake of Cd. Kido et al. reported that there existed some association between Cd-polluted rice intake and urinary Cd level and proteins of kidney dysfunction [12]. They concluded that the prevalence of β_2-microglobulin levels over 1 mg/g creatinine was higher in people who had eaten Cd-polluted rice than in those who had not. The mean urinary Cd concentration was statistically different between the two groups.

Nagata et al. studied male subjects 50–59 years of age living in a Cd-polluted area in Annaka City in 1987 [15]. The geometric mean value and standard deviation of urinary Cd excretion were 3.10 and 2.06 μg/g creatinine, respectively. In our study, these values were 2.20 and 1.87. If the data of Itahana are excluded, the values become 2.62 and 1.87 ($n = 13$).

There is a disagreement of opinion on the sensitivity of NAG activity for the Cd body burden [1, 2, 10]. It may partly derive from the analytical procedure such as age adjustment or use of nephrotoxic prevalence. The main problem seems to arise from the difference of urinary Cd levels. We calculated partial correlation coefficients after separating men and women to exclude the contribution of age and sex. The stratified results were the same as the combined ones. In addition, the prevalence of increased NAG activity over a set threshold was compared among the five districits. There was a statistical difference, but Ryan's multiple comparison could not be applied, which was in concordance with the result of the average comparison. Though the prevalence of NAG activity over a reference value was not sensitive, the age-adjusted correlation coefficients between Cd and NAG were significant both in men and women, which would support the usefulness of NAG in estimating low levels of Cd body burden.

A report of Cd pollution in the UK was recently published [24]. It gives the effect of smoking on the Cd body burden and that the urinary Cd excretion was little affected compared with the blood Cd level [25], which was the same result as in the present study. Their survey included the Cd content in the soil and dust, daily intake, and also the health effect, though their body burden of Cd is about a half that of districts in this study. We recommend a survey on the minimum health effects using the urinary Cd level to assess the amount of body burden and urinary specific protein including NAG, β_2-microglobulin, etc.

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