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Association of cadmium in urine and blood with age in a general population with low environmental exposure

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HIGHLIGHTS

- Lifetime variation of urinary Cd is similar to kidney Cd, after appropriate adjustment.
- Urinary Cd is significantly affected by blood Cd and urinary creatinine.
- Urinary Cd and blood Cd were not significantly higher in former smokers than never smokers.

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ABSTRACT

A recent study reported a nonlinear and nonmonotonic relationship between urinary cadmium (U-Cd) and age and questioned the long-held view that U-Cd is a reliable biomarker of Cd body burden at low exposure levels. In order to reassess the significance of U-Cd as biomarker of Cd body burden, we studied the lifetime trend of U-Cd as functions of diuresis in a cross-sectional study. Cadmium was measured with an inductively coupled plasma mass spectrometer (ICP-MS) for the general population taking part in the Metals and Health Survey in Jiangsu (MHSJ), China, with ages ranging from 2.8 to 86.8 years ($n = 1235$). Variations in U-Cd and B-Cd with age were modeled using natural cubic splines. Factors associated with U-Cd were analyzed with Pearson correlation and linear regression models. As results, nonsmoking men had peak U-Cd at approximately 60 years, after which it decreased. In nonsmoking women, U-Cd increased from 2.8 years to 50 years, then leveled off. In both genders, B-Cd increased from birth to approximately 30 years and then leveled off. U-Cd, expressed in per liter, was consistently associated with B-Cd and U-creatinine, regardless of smoking status. U-Cd and B-Cd were not significantly higher in former smokers than never smokers. Our study suggests that individual U-Cd level are correlated with B-Cd and U-creatinine, and needed to be appropriately adjusted for B-Cd and U-creatinine, when it is used for a biomarker of kidney burden of Cd.

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1. Introduction

Exposure to cadmium, a heavy metal present in cigarettes and enriched in rice, is an important public health issue in China and other countries (Wright et al., 2010; Lee et al., 2012; Zhang et al., 2014). Health risk assessments for Cd exposure in current environmental levels have become a critical focus of environmental health studies. The choice of an appropriate biomarker is the first step of risk assessments. Urinary cadmium (U-Cd) and blood cadmium (B-Cd) are the most frequently used biomarkers for the body burden of cadmium (Nordberg et al., 2012). U-Cd is mainly used for long-term exposure, while B-Cd measures recent exposure, with the two parameters having noticeable overlap (Adams and Newcomb, 2013). Several studies based on the premise that U-Cd reflects Cd body burden have been conducted over the past few years among the general population (Jarup et al., 2000; Nordberg,

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2010). However, many recent studies challenge this long-held view. The study by Chaumont et al. (2013) states that at low Cd exposure levels, U-Cd and age are associated through nonlinear and non-monotonic relationships that appear to be driven mainly by recent Cd intake and physiological variations in the excretion of creatinine and proteins. Another study shows a very high variation of U-Cd (adjusted for urinary creatinine, specific gravity, or flow rate) in individual urinary samples collected in 2 separate days at 6 fixed times over 24 h, and the authors conclude that associations between short-term changes in U-Cd and markers of kidney function within individual nonsmoking study participants are unlikely to reflect effects of Cd toxicity (Akerstrom et al., 2013). However, there have been different opinions on these studies (Adams and Newcomb, 2013), because no better biomarkers can substitute for the currently used U-Cd or B-Cd. In addition, the reported higher levels of U-Cd in children than in adolescents in Chaumont's study (Chaumont et al., 2013) is not consistent with previous reports on populations from the USA (USCDC, 2009) or Canada (Health-Canada, 2010). Therefore, it's necessary to reassess the significance of U-Cd as biomarker of Cd body burden.

B-Cd is another widely accepted biomarker for cadmium body burden. Adams and Newcomb (2013) previously stated that each 1% higher geometric mean U-Cd was associated with 0.50% higher B-Cd. The study by Chaumont et al. (2013) also proposed that recent Cd intake (B-Cd) and physiological variations in the excretion of creatinine might affect the change pattern of U-Cd with the variation of age. However, few studies concerned the lifetime distribution of B-Cd, and as such, a comparison of the variations of U-Cd and B-Cd over a lifetime would be particularly interesting for determining the relative sensitivities of these two biomarkers for physiological changes occurring during development and aging (Chaumont et al., 2013).

The objective of the present study is to reassess the significance of U-Cd and B-Cd as biomarkers of Cd body burden or cumulative exposure. We compared the lifetime trend of U-Cd and B-Cd as a function of gender, and analyzed the factors associated with U-Cd in different age groups.

2. Materials and methods

2.1. Study site and populations

This study was a part of the Metals and Health Survey in Jiangsu (MHSJ), performed in Changshu city, which is located in east of Jiangsu province of China with a total area of 1264 Km^2, and a residential population of 1.5 million at the end of 2013. The study design have been reported previously (Wang et al., 2016). In brief, the study population in Changshu city was composed of local residents, who lived at least 2 years in their current addresses. A total of 1235 subjects signed the informed consent form and participated this study. To the best of our knowledge, there has been no Cd pollution sources in Changshu city in the past years, so we proposed that the differences among age groups corresponded to changes in a nonsmoker's lifetime.

2.2. Sampling and ethics

Sample collection was conducted from May 2013 to Jan 2014. Morning urinary samples (20 mL) were collected at 7:00–8:00 a.m. and stored in polyacrylamide bottles. Venous blood samples were obtained from the arm of each participant in the residential community by registered physicians. Blood samples (2 mL) were drawn into vacutainers containing K2 EDTA (BD, Franklin Lakes, NJ) and shipped on dry ice to the laboratory of the Jiangsu Provincial Center for Disease Prevention and Control (CDC) for analysis. The weight and height of each participant were recorded with the participants wearing light clothing and no shoes or hats. We designed a structured questionnaire to capture the information on age, gender and chronic disease.

The study design of this work was approved by the institutional review board of the department of environmental health, Chinese CDC. All adult participants provided written, informed consent. For children, the written informed consent were obtained from their guardians.

2.3. Measurement of metals in urine and blood

The Cd and lead (Pb) in the collected blood samples and the Cd in urine samples were measured with an inductively coupled plasma mass spectrometer (ICP-MS; Thermo fisher X-series 2, Houston, TX, USA), using a previously described operating method for U-Cd (Sun et al., 2014). For blood samples, whole blood samples were treated with 0.1% Triton X-100 and 1% HNO_3 (Guarantee reagent, Merck), as well as 1% ammonia solution, before lead and Cd concentration analyses. The digests were diluted to a final volume of 50 mL using Milli-Q water (Millipore, Milford, MA, USA) and filtered through 0.45-μm filters for metal concentration. The metal concentrations in all samples were measured by ICP-MS. The limit of detection was 0.025 μg L^{-1} for B-Cd, 0.75 μg L^{-1} for B-Pb, and 0.02 μg L^{-1} for U-Cd. A total of 25 (25/1235) subjects had B-Cd below the limit of detection, and they were assigned a value representing the level of detection divided by the square root of 2 (Hornung and Reed, 1990). For internal quality assurance and control, standard reference materials were obtained from Seronorm™ trace elements whole blood level-1 (SERO, Norway). We measured Cd in the reference materials as 0.34 μg L^{-1}, felled into the certified ranges of 0.32–0.40 μg L^{-1}. We measured urinary creatinine (U-creatinine) with the kinetic Jaffe method (Slot, 1965) using an auto-analyzer (model 7180; Hitachi, Tokyo, Japan).

2.4. Statistical analysis

All biological parameters are reported as the median and interquartile range (IQR) and were log-transformed to an approximate normal distribution. U-Cd and other biomarkers were expressed per liter of urine and per gram of creatinine. We categorized the subjects into three groups, children (age < 12), adolescents (age: ≥12 and <18) and adults (age ≥ 18). The adult group was also further divided into 6 groups by age (as <30, <40 and decades intervals to <80, and ≥80). U-Cd in each age group was compared by ANOVA analysis, with covariables including gender, age, B-Cd, B-Pb and U-creatinine. The log U-Cd was compared across gender, with log U-creatinine as a covariable, by ANOVA followed by least squared means post hoc test.

Variations in U-Cd, B-Cd and U-creatinine with age were modeled using the natural cubic spline function. We assessed the relationship between log U-Cd and some independent variables, including sex, age, log B-Cd, log B-Pb, log U-creatinine and BMI, using Pearson's correlation and a general linear regression model in SAS. First, we used Pearson's correlation to select variables associated with Log U-Cd. Then, we applied stepwise forward selection to select covariables with a significance of 0.25 for a variable to enter and 0.10 to stay in the model. The condition index >10 was used as an indicator of the multi-collinearity. The underlying statistical assumptions about the homoscedasticity and normality of the errors were verified visually with regression residuals. Independence of the residuals was assessed by the Durbin-Watson test.

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