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Cadmium and mercury exposure over time in Swedish children

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ABSTRACT

Purpose: Knowledge about changes in exposure to toxic metals over time remains very sparse, in particular for children, the most vulnerable group. Here, we assessed whether a reduction in environmental pollution with cadmium (Cd) and mercury (Hg) caused a change in exposure over time. In total, 1257 children (age 4–9) in two towns in Sweden were sampled once in 1986–2013. Blood concentrations of Cd (b-Cd; n=1120) and Hg (b-Hg; n=560) were determined.

Results: The median b-Cd was 0.10 (geometric mean 0.10; range 0.010–0.61) μg/L and b-Hg was 0.91 (geometric mean 0.83; range 0.021–8.2) μg/L. Children living close to a smelter had higher b-Cd and b-Hg than those in urban and rural areas. There was no sex difference in b-Cd or b-Hg, and b-Cd and b-Hg showed no significant accumulation by age. b-Cd decreased only slightly (0.7% per year, $p < 0.001$) over the study period. In contrast, b-Hg did show a clear decrease over the study period (3% per year, $p < 0.001$).

Conclusions: The exposure to Cd was very low but still might increase the risk of disease later in life. Moreover, b-Cd only showed a minor decrease, indicating that Cd pollution should be further restricted. b-Hg was relatively low and decreasing, probably because of reduced use of dental amalgam and lower Hg intake from fish. The b-Cd and b-Hg levels decreased much less than the levels of lead in the blood as previously found in the same children.

1. Introduction

Many populations experience exposure to toxic metals at levels that cause toxic effects. Assessing risk and prioritizing preventive actions require information on the levels of exposure and their trends over time, particularly to evaluate the effectiveness of any actions taken. However, except for lead, which has been extensively monitored in several countries (Skerfving and Bergdahl, 2014), there is little information on exposure over time for important toxic metals, including two of the most relevant ones, cadmium (Cd) and mercury (Hg). These data are most notably lacking for children, a particularly vulnerable group.

Cd is a ubiquitous element and Cd exposure mainly occurs through food, and through smoking in adults (Nordberg et al., 2014). Cd may cause toxic effects on the proximal tubuli of the kidney (Åkesson et al., 2005; Suwazono et al., 2006) and skeleton (Åkesson et al., 2006, 2014), even at exposure levels that are present in the general population, and even in areas, such as Sweden, that have low environmental exposure, compared with most other sites worldwide (WHO, 2007). Women accumulate more Cd than men, making them more vulnerable to these effects. Furthermore, low to moderate levels of exposure to Cd during pregnancy may impair child growth (Kippler et al., 2012a; Johnston et al., 2014), kidney development (Hawkesworth et al., 2013), and cognitive function during early childhood (Ciesielski et al., 2012; Kippler et al., 2012b).

Cd exposure lends itself well to biomonitoring (Nordberg et al., 2014). The Cd concentration in blood (b-Cd) provides a combined index of recent exposure and the body burden. The body burden of Cd has extremely slow turnover, which attenuates any change in Cd uptake. Thus, the blood concentrations in adults may, to a large extent, reflect exposure from decades ago (Welinder et al., 1977; Liu et al., 2001). Hence, b-Cd in children is likely to be a valid biomarker for monitoring trends in exposure over time. However, in spite of the impact of Cd on public health, we have remarkably little information on the trends of any biomarker of Cd exposure over time; moreover, the information usually covers only short time periods, and is often inconclusive. The U.S. has been reporting b-Cd biomonitoring data since 1999–2000 as part of the NHANES/National Report on Human Exposure to Environmental Chemicals

Abbreviations: b-Hg, blood-mercury concentration; b-Cd, blood-cadmium concentration; b-Pb, blood-lead concentration

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(Ferraro et al., 2012) and there is some further information on b-Cd in adults (Wennberg et al., 2006; Schulz et al., 2007; Wiesmüller et al., 2007; Cerna et al., 2012; Baeyens et al., 2014). Still, there is almost no information on time trends in children (Schulz et al., 2007), especially young ones, partly because their b-Cd levels are extremely low and earlier studies primarily focused on health effects in adults.

Hg is also a ubiquitous element and Hg exposure mainly comes from food as organic methylmercury (MeHg) from fish, in particular from Hg-contaminated waters and large marine predatory species (Berlin et al., 2014). MeHg may cause toxic effects on the developing central nervous system (WHO, 2010; Berlin et al., 2014), and probably also causes coronary heart disease (Wennberg et al., 2012). Hg exposure occurs as well from dental amalgam fillings (Berlin et al., 2014). In this case, the exposure is to inorganic Hg, in particular elemental Hg vapour (Hg°), which evapourates from the surface of the filling. Hg° passes the blood-brain barrier, and may cause toxic effects; the toxic threshold is not known (Berlin et al., 2014). Hg exposure is also well suited for biomonitoring (Berlin et al., 2014). The Hg concentration in blood (b-Hg) provides a combined index of MeHg and Hg° exposure during the last months. In spite of the obvious relevance to public health, there is remarkably little information on the trends in Hg exposure over time. As with Cd, there are sparse data measuring exposure (as b-Hg and urinary Hg) and mainly in adults. The U.S. has been reporting blood Hg since 2003–2004 as part of the NHANES/National Report on Human Exposure to Environmental Chemicals, but there is almost no information on time trends in children.

We here report b-Cd from 1986–2013, and b-Hg from 1990–2013, as measured in large groups of children living in two towns in southern Sweden. These children have participated in long-term monitoring of blood-lead concentrations (b-Pb; children sampled yearly since 1978; Strömberg et al., 1995, 2003, 2008; Skerfving and Bergdahl, 2014; Skerfving et al., 2015). The b-Cd and b-Hg data have not been analysed and reported before.

2. Material and methods

2.1. Study area

Two towns in southern Sweden were studied (Strömberg et al., 1995). Landskrona (population 39,801 in mid-2006) has a secondary smelter established in 1944, which mainly handles scrap car batteries, but also melts other types of scrap, which may cause limited emissions of Cd and Hg (Landskrona Environmental management, 2014). The smelter is located about 1 km from the town centre and annually extract lead from approximately four million car batteries and about 70,000 t of other types of lead-containing batteries. There are no homes within 0.5 km from the smelter. The town of Trelleborg (population 40,136) has no metal-emitting industries.

2.2. Subjects

All children in the selected school classes of 1st to 3rd year (generally 7–10 years of age) were invited, and about 60% participated. Each year of sampling, different children were invited and sampled each year (cross-sectional samples). Also, in 1986, 53 preschool children (4–7 years of age) from Landskrona participated. The children did a structured interview and trained nurses did the blood sampling. The interview and the blood sampling took place at the schools.

In total, 1257 children were studied in 1986–2013 (one sample was analysed in each child). Children from Landskrona were sampled in 1986, 1990, 1991, 2004, 2006, 2007, 2009, 2011, and 2013; children in Trelleborg were sampled in 1991, 2003, and 2005. b-Cd (n=1120) and b-Hg (n=560) were determined.

The children in Landskrona were divided in three subgroups: (1) those living 0.5–1 km from the smelter, (2) other urban children, and (3) children living in rural areas. In Trelleborg, they were divided into urban and rural children. Information on parental smoking habits was obtained through a questionnaire.

The studies were approved by the Ethics Committee at Lund University; informed oral (children) and written (parents) consents were given.

2.3. Sampling

One blood sample (4 ml) was obtained from each child. Blood was retrieved from a cubital vein into heparinized evacuated tubes with no detectable amounts of Cd or Hg (Venoject VT-100SH; Treumo Europé, Leuven, Belgium 1986–2006 and Vacuette 4 mL; Lithium Heparin; Greiner-Bio One GmbH, Frickhausen, Germany 2007–2013). The samples were refrigerated until analysis. All samples collected in one year were analysed in duplicate in one experiment.

2.3.1. Cadmium determinations

All determinations were made in our laboratory. Three different analytical methods were employed at different times:

1986: Samples were analysed by electrothermal atomic absorption spectrometry (ETAAS, Perkin–Elmer HGA500/AA5000) and a deuterium background correction (Willers et al., 1988). In 0.5 mL blood, the proteins were precipitated by the addition of 0.5 ml deionized water and 0.5 ml ultrapure nitric acid. The technique of standard addition was used and included the addition of two different concentrations to each sample. The limit of detection (LoD; three times the standard deviation for the blank) was 0.05 μg/L and the method imprecision, calculated as the coefficient of variation (CV) for duplicate preparations measurements of 30 samples, was 18%. The accuracy was checked by analysis of external quality control samples (Supplementary Table S1).

1991: Samples were analysed by ETAAS (Varian Spectra AA-40; 283.3 nm; Zeeman background correction). One mL blood was deproteinized by addition of 2 mL of 1.4 m nitric acid (Stoeppler and Brandt 1980, slightly modified). Each sample was prepared in triplicate and Cd standard was added to one of the triplicate samples before deproteinization. Two injections (30 μL) of each sample preparation were made. The LoD was 0.01 μg/L and the CV calculated from samples without standard added was 11% in the range 0.08–0.30 μg/L, and 5% in the range 0.31–0.88 μg/L. The accuracy was checked by analysis of external quality control samples (Supplementary Table S1).

2003–2013: Inductively coupled plasma-mass spectrometry (ICP-MS; Thermo X7, Thermo Elemental, Winsford, UK) was employed. The sample were diluted ten-fold with an alkaline EDTA solution, according to Bárány et al. (1997). The Cd content was quantified in peak-jumping mode, using 114 Cd corrected for the spectral overlap of tin, 115Indium was used as an internal standard. The LoD, calculated as three times the standard deviation for the blanks, was 0.01–0.03 μg/L. All samples were prepared in duplicate and the method imprecision (CV) ranged 7.9–20%. Quality control (QC) samples were analysed along with the samples in each analysis year (Supplemental Table S1). In 2003–2007, we participated in the United Kingdom External Quality Assessment Service, with good agreement between obtained element concentrations in quality control samples used and expected values (Supplementary Table S2).

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