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Full length article Environmental and dietary exposure of young children to inorganic trace elements

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ABSTRACT

Children are exposed to toxic metals and metalloids via their diet and environment. Our objective was to assess the aggregate chronic exposure of children aged 3–6 years, living in France, to As, Cd, Cr, Cu, Mn, Pb, Sb, Sr, and V present in diet, tap water, air, soil and floor dust in the years 2007–2009. Dietary data came from the French Total Diet Study, while concentrations in residential tap water, soil and indoor floor dust came from the 'Plomb-Habitat' nationwide representative survey on children's lead exposure at home. Indoor air concentrations were assumed to be equal to outdoor air concentrations, which were retrieved from regulatory measurements networks. Human exposure factors were retrieved from literature. Data were combined with Monte Carlo simulations. Median exposures were 1.7, 0.3, 10.2, 34.1, 60.3, 0.7, 0.1, 44.3, 1.5 and 95th percentiles were 4.4, 0.5, 15.8, 61.3, 98.3, 2.5, 0.1, 111.1, 2.9 µg/kg bw/d for As, Cd, Cr, Cu, Mn, Pb, Sb, Sr, and V respectively. Dietary exposures dominate aggregate exposures, with the notable exception of Pb - for which soils and indoor floor dust ingestion contribute most at the 95th percentile. The strengths of this study are that it aggregates exposures that are often estimated separately, and uses a large amount of representative data. This assessment is limited to main diet and residential exposure, and does not take into account the relative bioavailability of compounds. These results could be used to help target prevention strategies.

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1. Introduction

Many metals are known to have adverse impacts on human health via various toxicological mechanisms. These have also been subject to exposure surveillance indicating that reference values may be reached for certain populations – for instance for lead, cadmium and Arsenic (WHO, 2015). This is particularly true of lead: in France in 2008–2009, for example, more than half of young children had blood lead levels above the no observed effect level (Etchevers et al., 2014). Because they are subject to developmental toxicity (Grandjean et al., 2008) and because their lower body weight and more intense hand to mouth activity means they are most exposed, children are of particular concern. Metals and metalloids are widely distributed and ubiquitous in the environment, so that people are exposed via both food and environment.

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Biomonitoring studies are ideal for taking into account all sources of exposure without allowing source apportionment, while indirect methods modeling doses from contamination and consumption data enable estimation of the specific contribution made by a given exposure medium (Lioy and Rappaport, 2011), which is useful with a view to prevention measures. However, given the amount of data that are required, many indirect exposure assessments are dedicated to one particular source or media (diet, water, soil, dust...) of exposure. This is true of total diet studies (TDS), designed to assess dietary exposure to many compounds, as well as studies designed to address a particular environmental situation such as an industrial setting. The consequence is that there is often a choice to be made between knowledge of overall, or aggregate, exposure, and awareness of the significance of different media and sources of exposure. The result is that there are very few indirect exposure assessments taking into account most pathways of exposures for the general population, and then quantifying the relative importance of each one. Aggregate exposure assessment allows such a global view, which could reveal useful in regulatory context for setting a media standard accounting for others.





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In the present study, our objective is twofold. It is to assess, for 3–6 year-old children living in France, firstly aggregate chronic exposure to arsenic (As), cadmium (Cd), chromium (Cr), copper (Cu), manganese (Mn), lead (Pb), antimony (Sb), strontium (Sr) and vanadium (V) and secondly, to identify the main contributors to exposure to these toxic metals and metalloids. We focused on the general population, living aside particular situations such as local contamination or specific consumption habits. The ultimate goal is to help identifying the relevant pathways of exposure for young children, which could be useful for setting dietary or environmental standards.

We chose these metals because of their toxicity and because their contamination data were available at nationwide scale, for both food and residential environment. Other environments such as public living places were not included in the assessment.

2. Material and methods

2.1. Assumptions

We calculated external exposures, assuming a total absorption of contaminant by inhalation and ingestion pathways. We did not take into account the dermal route, since this is assumed to be negligible for inorganic metals and metalloids (Nordberg et al., 2014). Children's environmental exposure was assumed to be equivalent to residential exposure, i.e. at home where they spend the largest amount of time. As indoor air data were not available, indoor air concentrations were assumed to be equivalent to outdoor air concentrations. Dietary exposure was limited to food items included in the French Total Diet Study (Anses, 2011). As we focused on chronic exposures, we did not take into account the temporal variability of exposures. In order to give an idea of the possible risk for children, a comparison is made when possible, between the aggregate exposure and an international tolerable daily intake (TDI). This comparison is to be taken with care, because a total absorption was supposed whatever considered route of exposure.

2.2. Exposure model

We aggregated external chronic exposures via oral (ingestion) and respiratory (breathing) routes by summing them in a probabilistic framework. Exposure of individual i to contaminant j was calculated using Eq. (1).

$$E_{i,j} = Eresp_{i,j} + Eing_{i,j}$$
(1)

With $E_{i,j}$: daily exposure of individual i to contaminant j, $Eresp_{i,j}$: daily respiratory exposure of individual i to contaminant j (µg of contaminant/kg bw/d), $Eing_{i,j}$: daily ingestion exposure of individual i to contaminant j (µg of contaminant/kg bw/d).

Respiratory exposure was calculated using Eq. (2).

$$Eresp_{i,j} = \frac{C_j \times V_i}{BW_i}$$
(2)

With V_i: total daily respired volume of individual i (m^3/d) , C_j: air concentration of contaminant j (µg contaminant/m³), BW_i: body weight of individual i (kg bw).

Ingestion exposure was calculated using Eq. (3).

$$\operatorname{Eing}_{i,i} = \operatorname{Ediet}_{i,i} + \operatorname{Ewater}_{i,i} + \operatorname{Edust}_{i,i} + \operatorname{E}\operatorname{soil}_{i,i}$$
(3)

With Ediet_{i,j}: dietary exposure of individual i to contaminant j (µg contaminant/kg bw/d), Ewater_{i,j}, Edust_{i,j}, and E soil_{i,j}: exposure of individual i to contaminant j in drinking water (tap), dust and soil, respectively (µg contaminant/kg bw/d).

Dietary exposure was calculated using Eq. (4).

$$\mathsf{Ediet}_{i,j} = \frac{\displaystyle\sum_{k=1}^{n} Q_{k,i} \times C_{k,j}}{\mathsf{BW}_{i}} \tag{4}$$

With $Ediet_{i,j}$: dietary exposure of individual i to contaminant j (µg contaminant/kg bw/d), n: number of food items in the diet, $Q_{k,i}$: food k consumption by individual i (g food/d), $C_{k,j}$: concentration of contaminant j for food k (µg contaminant/g food).

Tap water exposure was calculated as follows, using Eq. (5). NB: bottled water was treated as a food item.

$$\mathsf{Ewater}_{i,j} = \frac{\mathsf{Q}_{\mathsf{w}i} \times \mathsf{C}_j}{\mathsf{BW}_i} \tag{5}$$

With Ewater_{i,j}: tap water exposure of individual i to contaminant j (μ g contaminant/kg bw/d), Qw_i: tap water consumption of individual i (L/d), C_j: tap water concentration in contaminant j (μ g contaminant/L). Exposure to dust was calculated as follows, using Eq. (6).

$$Edust_{i,j} \frac{\frac{C_{s,j}}{L_s} \times Q_{di}}{\frac{BW_i}{BW_i}}$$
(6)

With Edust_{i,j}: dust exposure for individual i to contaminant j (µg contaminant/kg bw/d), Q_{di}: dust ingestion rate for individual i (mg dust/d), C_{s,j}: surface s concentration of contaminant j (µg contaminant/m²), L_s: dust loading of surface s (mg dust/m²).

Exposure to soil was calculated as follows, using Eq. (7).

$$\mathrm{Es}_{i,j} = \frac{\mathrm{Q}_{\mathrm{s}i} \times \mathrm{C}_{j} \cdot 10^{-3}}{\mathrm{BW}_{\mathrm{i}}} \tag{7}$$

With Esoil_{i,j}: soil exposure for individual i to contaminant j (µg contaminant/kg bw/d), Q_{si} : soil ingestion rate for individual i (mg dust/d), C_i : soil concentration of contaminant j (µg contaminant/g of soil).

2.3. Human exposure factors: consumption, intake rates, body weights and dust loadings

The food and water consumption of children aged 3–6 years was retrieved from the corresponding 244 individual data sets from the French 'INCA2' representative food consumption survey, which recorded food habits over a period of seven consecutive days (Lioret et al., 2010). Body weight data were also available from this survey. Total daily respired volumes, as well as dust and soil intake rates, were retrieved from the exposure factors handbook (EPA, 2011). Mean and 95th percentiles were 10.1, 13.8 m³ (air)/d; 60, 100 mg (dust)/d; 50, 200 mg (soil)/d. We used the normal (left truncated to zero) shapes of distributions indicated in the exposure factors handbook (lognormal for dust intake as stated by Ozkaynak et al., 2011). Dust loading was estimated by weighting geometric means and standard deviation indicated in (Giovannangelo et al., 2007), and resulted in 255.3 mg (dust)/m² with geometric standard deviation of 3.23 mg (dust)/m².

2.4. Food and environment contamination data

Food contamination data are from the last French Total Diet Study: the most consumed (consumer rate of at least 5%) foods identified in consumer survey (Lioret et al., 2010) plus the main known or assumed food contributors to exposure were selected (Anses, 2011). It led to 212 food items of 41 food groups covering around 90% of the whole diet (Arnich et al., 2012). 19,830 of these food items were sampled in eight metropolitan regions, over two different seasons, between June 2007 and January 2009 in order to reflect chronic exposure. Before analysis,

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