

Concentrations of Environmental Chemicals in Urine and Blood Samples of Children from San Luis Potosí, Mexico

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Abstract Human biomonitoring (HBM) is an appreciated tool used to evaluate human exposure to environmental, occupational or lifestyle chemicals. Therefore, the aim of this study was to evaluate the exposure levels for environmental chemicals in urine and blood samples of children from San Luis Potosí, Mexico (SLP). This study identifies environmental chemicals of concern such as: arsenic (45.0 ± 15.0 µg/g creatinine), lead (5.40 ± 2.80 µg/dL), t,t-muconic acid (266 ± 220 µg/g creatinine), 1-hydroxypyrene (0.25 ± 0.15 µmol/mol creatinine), PBDEs (28.0 ± 15.0 ng/g lipid), and PCBs (33.0 ± 16.0 ng/g lipid). On the other hand, low mercury (1.25 ± 1.00 µg/L), hippuric acid (0.38 ± 0.15 µg/g creatinine) and total DDT (130 ± 35 ng/g lipid) exposure levels were found. This preliminary study showed the tool's utility, as the general findings revealed chemicals of concern. Moreover, this screening exhibited the need for HBM in the general population of SLP.

Keywords 1-OHP · Benzene · DDT · Metals · PBDEs · PCBs

Humans are exposed to a diversity of chemical compounds due to natural and/or anthropogenic activities that seriously contaminate environmental compartments such as: water, air, soil, dust and sediment (Suk et al. 2016). Also, some chemical compounds can be entering the food chain, leading to an increased exposure of people through ingestion of contaminated foods. Moreover, several acute and chronic adverse health effects on the brain, liver, kidney, cardiovascular system, among others, have been documented following exposure to environmental chemicals (Rich 2017). It has therefore been suggested that human exposure assessment to environmental chemicals is a crucial step towards the prevention of chemical-induced illnesses in vulnerable populations (Trejo-Acevedo et al. 2009).

In this regard, human biomonitoring (HBM) has emerged as an important tool to identify and quantify exposure levels to environmental chemicals in order to contribute to the development of strategies and programs to protect human health (NHANES 2009). HBM studies are aimed at the quantification of chemical compounds and/or their metabolites in biological samples such as blood, urine, hair and/or milk (NHANES 2009). Although an increasing number of HBM studies worldwide have been performed, in developing countries such as Mexico, information about human exposure to environmental chemicals is very limited, because HBM studies have not been established. Current reports suggest that children appear to be particularly suitable for HBM studies, as they are not occupationally exposed. Moreover, children are more sensitive than adults to adverse health effects exerted by environmental chemicals (Armstrong et al. 2002). Also, children absorb more from their surroundings than adults, even when exposed to the same concentrations of environmental contamination (Armstrong et al. 2002).

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Therefore, the aim of this study was to evaluate the exposure profile for arsenic (As), lead (Pb), mercury (Hg), 1-hydroxypyrene (1-OHP, exposure biomarker for polycyclic aromatic hydrocarbons), hippuric acid (HA, exposure biomarker for toluene), trans, trans-muconic acid (t,t-MA; exposure biomarker for benzene), total DDT [1,1-bis(p-chlorophenyl)-2,2,2-trichloroethane (DDT) and 1,1-dichloro-2,2-bis(p-chlorophenyl) ethylene (DDE)], polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs); in urine and blood samples of children from San Luis Potosí, Mexico.

Materials and Methods

Children participating in the study were recruited from the city of San Luis Potosí (SLP) (an urban site with high vehicular traffic, located near a metallurgical industry, with previous reports of inorganic arsenic groundwater contamination; Diaz-Barriga et al. 1993) between 2015 and 2016.

After delivering a personal conference with the parents at the area of study, 55 children attending public school, between the ages of 6–12 years were included in the analysis. The recruitment strategy for children was previously reported by Ochoa-Martinez et al. (2016). A signed informed consent was collected from the parents before urine and blood samples were taken. Also, anthropometric measures (weight, height, body mass index) of all children incorporated in this research were obtained. The body mass index (BMI) was estimated using the following equation: $BMI = \text{body weight (Kg)} / [\text{body height (m)}]^2$.

Furthermore, characteristics such as: age, sex, smoking status (active and/or passive), household and sociodemographic characteristics, the occupation of family members, among others, were obtained through the application of a short survey. Finally, collection of blood and urine sample was performed according to procedures previously described by our work group (Ochoa-Martinez et al. 2016). Briefly, approximately 50–100 mL of urine (the first-morning urine) were collected in sterile plastic cups. Then, aliquots (2 mL) of the urine were made in sterile polypropylene tubes and immediately placed on ice. Blood samples were drawn after overnight fasting (from the cubital vein) inside tubes without anticoagulant for serum collection (serum was obtained after blood samples were centrifuged at 1200xg for 10 min), serum samples were transferred to hexane-rinsed brown glasses. Also, blood samples were obtained and stored in tubes with anticoagulant (ethylenediaminetetraacetic acid). Urine, blood, and serum samples were transported to the laboratory and stored at -80°C until analysis. Blood and serum samples were used to determine PBDEs, PCBs, total DDT and lead levels. Whereas, urine samples were used to evaluate As, Hg, t,t-MA, HA,

and 1-OHP concentrations. The Biomedical Ethics Committee from the Faculty of Medicine of the Autonomous University of San Luis Potosí reviewed and approved the study protocol.

All the assessed chemical compounds were quantified using analytical methods previously developed and described by our research group (Ochoa-Martinez et al. 2016). For quality control purposes, the following certified standards were used: organic contaminants in fortified human serum (NIST SRM 1958) for DDTs, PCBs, and PBDEs; IRIS Clin Cal Recipe (50013, 8867 and 50014) for 1-OHP; NIST SRM 2670 for urinary arsenic; CDC.WS2H proficiency testing blood material, method codes 3851 for blood lead; IRIS Clincheck D-80335 for urinary mercury, and IRIS Clin Cal Recipe 9969 for HA and t,t-MA. The average recovery was between 90%–110% for all compounds tested.

Descriptive statistics [median (PC50), geometric mean, standard deviation, minimum, maximum, percentiles] were calculated from raw data for all evaluated biomarkers of exposure.

Results and Discussion

Table 1 shows the general characteristics of children (boys and girls) participating in this study. Ranging from 6 to 13 years of age and the mean value for age was 9.50 ± 4.00 years (geometric mean \pm standard deviation). Approximately 60% of the children participating in the study were girls. The mean value for height was 130 ± 16.5 cm, for weight the values ranged from 20.5 to 45.0 Kg (28.5 ± 7.50).

Regarding the level of urinary metals (Table 2), a mean level of 47.5 ± 7.00 $\mu\text{g/g}$ of creatinine, and 1.25 ± 1.00 $\mu\text{g/L}$ were found in the assessed urine samples for arsenic and mercury, respectively. Although, mean levels of urinary arsenic found in this study were lower than 50 $\mu\text{g/g}$ of creatinine (the Center for Diseases Control and Prevention in the USA (CDC) action level) (NHANES 2009), approximately 15% (data not shown) of the children assessed had concentrations of urinary arsenic (UAs) above that CDC guide. When comparing UAs levels found in our research

Table 1 General characteristics of assessed population

Variable	Mean \pm SD (range)
Age (years)	9.5 ± 4.0 (6.0–13.5)
Boys (%)	40.0
Height (cm)	130 ± 16.5 (115–145)
Weight (Kg)	28.5 ± 7.50 (20.5–45.0)
Body mass index (BMI, kg/m^2)	18.0 ± 3.0 (12.0–23.0)

Table 2 Urinary metals, t,t-MA, HA, 1-OHP and blood lead concentrations of children living in San Luis Potosí, Mexico

Compound (unit concentration)	Mean ^a	SE ^b	PC25 ^c	PC50	PC75	PC95	min ^d	max ^e
Arsenic (µg/g of creatinine)	47.5	7.00	25.0	45.0	50.0	75.0	<LOD	87.5
Mercury (µg/L)	1.25	1.00	1.00	1.50	1.80	2.90	<LOD	11.5
Lead (µg/dL)	5.75	2.50	2.75	6.00	7.90	9.00	1.00	12.5
1-OHP (µmol/mol of creatinine)	0.25	0.15	<LOD	0.20	0.30	0.60	<LOD	0.75
t,t-MA (µg/g of creatinine)	280	195	150	225	350	845	35.0	850
HA (g/g of creatinine)	0.38	0.15	0.10	0.35	0.65	1.15	0.10	1.75

Detection limit (LOD) for arsenic = 2.5 µg/g creatinine, LOD for mercury = 2 ng/L, LOD for lead = 1.0 µg/dL, LOD for 1-OHP = 0.5 µg/L, LOD for t,t-MA and HA = 0.03 mg/mL

^aValues are geometric means

^bStandard Deviation (SD)

^cPercentile (PC)

^dMinimum (min)

^eMaximum (max)

with levels detected in the Fourth National Report on Human Exposure to Environmental Chemicals in the study done in the USA (NHANES IV) (children aged 6–11 years), we can observe a lower mean level in NHANES IV (8.25 µg/g creatinine). However, UAs concentrations of assessed samples were similar (an unexpected finding) to the levels found in the urine of children living in a mining site (an area contaminated with metals) located in SLP (45.0 ± 35.5 µg/g creatinine) (Jasso-Pineda et al. 2015) and higher than the values found in urine samples of children from Ciudad Juarez, Chihuahua, México (CJC) (an industrial site) (19.5 ± 17.0 µg/g creatinine) (Ochoa-Martinez et al. 2016). In this respect, previous studies have demonstrated environmental contamination (air, soil, household dust, and tap water) due to arsenic in the study area (Diaz-Barriga et al. 1993). Therefore, those contamination sources could explain the high exposure levels found in this research. Moreover, toxic effects, such as DNA damage, have been observed in children exposed to similar levels of urinary arsenic such as the ones found in this study (Jasso-Pineda et al. 2015). Regarding urinary mercury levels, 0.42 and 0.40 µg/L were found in the study performed by NHANES IV (children aged 6–11 years) and in the German Environmental Survey, respectively. For studies performed in Mexico, a urinary mean level of 0.90 µg/L was found in children living in Mexico City (Basu et al. 2014) and approximately 4.50 µg/L in urine from children recruited in mining sites (Costilla-Salazar et al. 2011). No noticeable exposure sources of mercury were observed in the studied area. Moreover, all urine samples assessed in this investigation had levels below 25.0 µg/L, urinary mercury concentrations above that value (25.0 µg/L) indicate an increased health risk (Schulz et al. 2007).

For 1-OHP, the levels ranged from <LOD to 0.75 µmol/mol of creatinine (0.25 ± 0.15 µmol/mol of creatinine) (Table 2). The urinary levels of 1-OHP found in this

work (an urban site with high vehicular traffic) are in line with urinary concentrations quantified (children aged 6–12 years) in a previous study performed in a community with high vehicular traffic in Mexico (mean value: 0.20 µmol/mol of creatinine) (Martínez-Salinas et al. 2010), but were lower than urinary 1-OHP concentrations found in children living in sites with high-risk of air pollution such as: rural communities that use biomass combustion as the main energy source (3.25 µmol/mol of creatinine; indoor air pollution), communities with brick kiln industries (0.35 µmol/mol of creatinine), and communities located next to a sanitary landfill (0.30 µmol/mol of creatinine) (Martínez-Salinas et al. 2010). For American children (6–11 years) included in the NHANES IV study, the mean level found was approximately 0.05 µmol/mol of creatinine, which is lower than the one found in this study (NHANES 2009). Although urinary 1-OHP levels detected in this study were lower than values found in other scenarios in Mexico, they (concentrations found in this investigation) have a significant potential for generating adverse effects on human health, as demonstrated in a previous research (Pruneda-Alvarez et al. 2016a). Also, urinary t,t-MA and HA levels found in our investigation are depicted in Table 2, for t,t-MA, the mean level detected was 280 ± 195 µg/g of creatinine, whereas for HA it was 0.38 ± 0.15 g/g of creatinine. The mean urinary t,t-MA level found in the samples analyzed in this work was higher than what was recorded in several studies performed in different scenarios worldwide (NHANES 2009; Protano et al. 2012; Kim et al. 2017). For example, Protano et al. 2012, assessed t,t-MA levels in urine samples of 396 Italian children (5–11 years) living in three areas with different degrees of urbanization, the mean urinary level was approximately 126 ± 123 µg/g of creatinine (Protano et al. 2012). However, slightly higher levels of urinary t,t-MA than the ones found in this work were detected in a petrochemical area in Mexico, where

a mean level of 374 $\mu\text{g/g}$ of creatinine was found in urine samples of children aged 6–12 years (Pelallo-Martínez et al. 2014). Regarding urinary HA levels, the urinary concentrations quantified in this work were also higher than what was shown in other studies (Kim et al. 2017), a mean urinary HA level of approximately 0.25 g/g of creatinine was found in Korean children (Kim et al. 2017). Important contributors to individual t,t-MA and HA exposure are motor vehicle exhausts, industrial emissions, domestic biomass combustion, consumption of products and environmental tobacco smoke, among others (Pruneda-Alvarez et al. 2016b). As shown above, high vehicular traffic and industrial emissions could be the main exposure sources for polycyclic aromatic hydrocarbons and volatile organic compounds in the researched area. Table 2 shows the levels of blood lead found in this study, where a mean level of 5.75 ± 2.50 $\mu\text{g/dL}$ was detected. That mean level is lower than the guide level of 10.0 $\mu\text{g/dL}$ established by the CDC in the USA (NHANES 2009) and only 5% of the assessed children had levels higher than that guide (data not shown). In addition, approximately 20% of the analyzed samples of blood lead concentrations were above 5.00 $\mu\text{g/dL}$ (data not shown). In this regard, the mean blood lead level found in the NHANES IV study was 1.50 $\mu\text{g/dL}$ for children aged 6–11 years (NHANES 2009), while in the German Environmental Survey on children which was a study that took place from 2003 to 2006, the reference value is 3.50 $\mu\text{g/dL}$ (Schulz et al. 2009), both levels (USA and Germany) are lower than concentrations of blood lead quantified in

evaluated samples in this study. The blood lead levels found in this investigation were expected, due to the presence of a metallurgical industry located near the study area. Moreover, blood lead levels found in this work are a concern, because toxic effects on central nervous system in children have been associated with blood lead levels below 10.0 $\mu\text{g/dL}$ (Lanphear et al. 2005).

For PCBs, we focused on three (PCB138, PCB153, and PCB180) out of the six PCB indicators proposed as markers of PCB contamination. Those congeners are three of the PCBs that are most frequently found in humans and the environment (Ferrante et al. 2014). The mean levels detected in the serum of children included in this work were 7.90 ± 2.00 , 13.51 ± 3.50 , 13.0 ± 4.00 , and 34.5 ± 10.0 ng/g lipid for PCB138, PCB153, PCB180, and total PCB indicators, respectively (Table 3). In this respect, the mean levels of the same congeners in NHANES IV study (age group: 12–19 years) were lower (4.97, 5.86, and 3.06 ng/g lipid, for PCB138, PCB153, and PCB180, respectively) (NHANES 2009). On the other hand, similar blood PCBs levels were observed when our results were compared with levels quantified in the Canadian Health Measures Survey (CHMS, males and females aged 6–79 years), finding approximate mean levels of 10.0, 18.5, and 15.0 ng/g lipid for PCB138, PCB153, and PCB180, respectively (Haines and Murray 2012). However, higher PCBs levels were reported in a biomonitoring study that evaluated blood PCBs levels in Inuit children of 11 years of age, for PCB138, a mean level of 35.7 ng/g lipid was found, regarding PCB153 and

Table 3 Serum persistent organic pollutants (PCBs, PBDEs and total DDT) concentrations of children living in San Luis Potosí, Mexico (ng/g lipid)

Chemical compound	Mean ^b	SD ^c	PC25 ^d	PC50	PC75	PC95	min ^e	max ^f
PCB 138	7.90	2.00	1.75	7.00	8.50	12.0	<LOD	15.5
PCB 153	13.5	3.50	3.50	15.0	19.0	25.0	<LOD	35.0
PCB 180	13.0	4.00	2.00	14.0	17.0	23.0	<LOD	29.0
Total PCB indicators	34.5	10.0	7.25	36.0	44.5	60.0	<LOD	79.5
PBDE 47	9.50	2.50	3.00	8.50	12.0	19.5	<LOD	25.0
PBDE 99	7.50	2.00	1.50	7.00	11.5	16.0	<LOD	22.5
PBDE 100	5.00	2.50	1.50	4.50	10.0	13.0	<LOD	20.0
PBDE 153	7.00	3.00	1.00	8.00	15.5	25.0	<LOD	50.0
PBDE 154	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
Total PBDEs	29.0	10.0	7.00	28.0	49.0	73.5	<LOD	118
^a Total DDT	130	35.0	27.5	120	155	255	17.5	410

LOD for all PCB congeners were approximately 0.5 ng/g lipid, LOD for all PBDE congeners were approximately 1.5 ng/g lipid, LOD for DDT and DDE were approximately 3.5 ng/g lipid

Blood concentrations are shown in ng/g lipid

^aSum of the levels of each congener analyzed (DDT and DDE)

^bValues are arithmetic means

^cStandard Deviation (SD)

^dPercentile (PC)

^eminimum (min)

^fmaximum (max)

PCB180, the mean blood levels were 73.6, and 32.5 ng/g lipid, respectively (Boucher et al. 2016). In Mexico, Ochoa-Martinez et al. 2016, assessed serum PCBs levels in children (aged 6–12 years) living in the CJC (Mexico)–El Paso (USA) area (the major manufacturing center in the Mexico–USA border, with important electronic companies established in that zone). The mean blood PCBs levels found in Ochoa-Martinez et al. 2016 study were slightly lower (6.75, 5.90, and 4.75 ng/g lipid for PCB138, PCB153, and PCB180, respectively) than the ones shown in this research (Ochoa-Martinez et al. 2016). In our study, the levels of total DDT found in analyzed samples ranged from 17.5 to 410 ng/g lipid (mean level of 130 ± 35.0 ng/g lipid) (Table 3). As was expected, the levels of total DDT detected in our analysis were lower than those previously reported in the blood of children living in endemic malaria regions of Mexico (range 2200–34,000 ng/g lipid), where DDT was used in agricultural and public health campaigns (Pérez-Maldonado et al. 2014). On the other hand, total DDT levels were found to be similar to the ones reported in NHANES IV (125 ng/g lipid) and CHMS (155 ng/g lipid) studies. No noticeable exposure sources were detected for DDT and PCBs. Furthermore, we can assume that the diet is the main pathway through which people living in SLP are exposed to both persistent organic pollutants (POPs). For these chemical contaminants (DDT and PCBs), it is problematic to define health risks, as guidelines to protect human health have not been recognized. Therefore, large studies are required to evaluate the potential human health risk for exposure to those chemicals in the evaluated area. Finally, the PBDEs levels in analyzed serum samples are shown in Table 3. Total PBDEs levels ranged from <LOD to 118 ng/g lipid, and the mean total PBDEs level was 29.0 ± 10.0 ng/g lipid. Moreover, according to previous studies done in North America (NHANES 2009), the principal congener found was BDE47 (9.50 ± 2.50 ng/g lipid), followed by BDE153 (7.00 ± 3.00 ng/g lipid), then by BDE99 (7.50 ± 2.00 ng/g lipid), and by BDE100 (5.00 ± 2.50 ng/g lipid), and finally by BDE154 with levels below the detection limit. The mean total PBDEs level found in NHANES IV (subjects aged 12–19 years) was approximately 50.0 ng/g lipid (NHANES 2009), in the CHAMACOS (Center for the Health Assessment of Mothers and Children of Salinas in California) study, children of 9 years of age had a mean level of total PBDEs (BDE47, BDE99, BDE100 and BDE153) of 63.1 ng/g lipid (Harley et al. 2017) and children (of 8 years of age) participating in the Health Outcomes and Measures of the Environment (HOME) Study, had a mean level of total PBDEs (BDE28, BDE47, BDE99, BDE100 and BDE153) of 46.3 ± 2.2 ng/g lipid (Vuong et al. 2017), as noted, the levels found in NHANES IV, CHAMACOS, and HOME studies are slightly higher than the ones found in this research.

In Mexico, a recent study that evaluated PBDEs levels in the blood of children living in CJC (Mexico)–El Paso (USA) area showed similar levels (29.5 ± 28.0 ng/g lipid) than the ones detected in children living in SLP (this study) (Ochoa-Martinez et al. 2016). Regarding congeners profile, the major metabolite found in all studies (NHANES IV, CHAMACOS, HOME, and this study) was BDE47. In this regard, the main component of the principal PBDEs mixture market in North America (penta-BDE technical mixture) was BDE 47 (Birnbbaum and Staskal 2004). The main source of PBDEs exposure would appear to originate from the intensive use of electrical applications at the assessed site (such as computers, cell phone, television, among others), as suggested in a previous study (Perez-Vazquez et al. 2015).

HBM of vulnerable populations (as assessed in this study) is a valuable strategy for the identification of critical contaminants, detection of high-risk populations, surveillance of the general population, among others (NHANES 2009). In this regard, although with proper precaution concerning the low number of analyzed samples in the current assessment, the evidence shown in this work was useful to identify environmental chemicals of concern for the assessed population such as: arsenic (since urinary arsenic levels detected in this study were similar to the ones found in contaminated sites with metals), lead (since the mean level in our research was higher than $5.0 \mu\text{g/dL}$), t,t-MA (this study detected levels of that metabolite slightly lower than the ones quantified in high-risk zones, such as a petrochemical area) PCBs (higher levels of three PCB indicators were found in our screening study compared to levels detected in an industrial area in Mexico), PBDEs (we found similar serum concentrations in assessed samples compared to blood levels detected in an industrial area in Mexico). Moreover, the data reported in this preliminary study in SLP suggests the need to perform an HMB study in the general population of SLP to determine baseline exposure to analyzed chemicals.

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