



Research Article

Prenatal Exposure to Arsenic and Body Mass Index Trajectories in Child: The POSGRAD Birth Cohort Study

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Abstract

Background: Early-life exposure to toxic metals may promote rapid postnatal weight gain and development of excess adiposity, though findings have been inconsistent across metal type and study. **Objective:** To evaluate the association between prenatal exposure to arsenic and body mass index (BMI) trajectories in participants of the POSGRAD birth cohort study. **Methods:** We included 450 mother-child pairs. Exposure to arsenic was evaluated during pregnancy and the children were followed from birth to 10 years of age. We use a General Equation Model (GEEs) to examine the association between the main variables. **Results:** The obesity in mothers prior to pregnancy was 2.2%. The BMI at 10 years was 19.4 (3.6). The arsenic concentrations were 0.06 mg/L (0.04mg/L). We found a significant association between prenatal exposure to arsenic and the BMI trajectories ($b_{\text{quartile2}} = 2.18$, $b_{\text{quartile3}} = 2.54$ vs quartile 1, $p < 0.05$). **Conclusions:** The study strengthens the evidence that BMI in children is associated significantly with prenatal exposure to arsenic.

Keywords: Prenatal exposure; Cohort study; Arsenic; BMI, Obesity

Introduction

Childhood overweight/ obesity is one of the most important public health problems worldwide and its prevalence continues to increase alarmingly [1]. Mexico is one of the countries most affected by this epidemic, with prevalence's of obesity in school-age children and adolescents of 33% and 36%, respectively

[2]. One of the most important implications of childhood overweight/obesity is that increased the risk of a wide variety of chronic degenerative diseases in adulthood, including diabetes, hypertension, ischemic heart disease, and lipid metabolism disorders, among others [3]. Excessive calorie intake, genetic factors and sedentary lifestyle are the main known risk factors for the development of obesity; however, not all cases are explained by these conditions [3-5]. In this sense, it has been reported that certain environmental determinants [6] are capable of modifying

the susceptibility to develop metabolic disorders, signalling, that exposure to environmental pollutants (even at low concentrations) called endocrine disruptors [7] may cause alterations in the control of the energy balance towards lipid production and accumulation for his ability to interfere with the normal functioning of natural hormones. The most studied compounds in recent years include phthalic acid esters, bisphenol A and heavy metals [7].

Of particular interest in this paper is to evaluate the prenatal exposure to inorganic Arsenic (iAs), a naturally occurring and abundant element in the environment that is found in groundwater and certain foods (e.g., rice, grains) [8]. It is estimated that 140 million people worldwide are exposed to arsenic-contaminated drinking water at levels above the World Health Organization's recommended guideline value [9]. In the United States, approximately 13 million Americans are exposed to arsenic from drinking water that exceeds the Environmental Protection Agency's current standard of 10 µg/L [10]. Mexico is one of the most affected countries, estimating that approximately six million inhabitants have an exposure to As especially naturally. Similarly, the state of Morelos is considered an area with significant concentrations of As, primarily due to mining activities [11]. Epidemiological studies identify As a risk factor for human health [10,12,13], until now and even with the vast number of studies performed [14]. It is not well known the molecular mechanisms of the toxic and carcinogenic effects of As. In addition, the number of metabolic pathways altered by As is too extensive to fully understand, and there is not a suitable animal model for the study of the long-term toxicity effects of As exposure. The toxicity of arsenic is influenced by its metabolism, after absorption, iAs is metabolized into mono- and di-methylated compounds (MMA and DMA) and the three arsenic forms are excreted in the urine [12]. iAs is a carcinogen and long-term exposure is known to cause skin lesions and carry an elevated risk of cancer, among other health effects, including cardiometabolic risk outcomes [13]. The association between arsenic exposure and obesity has been poorly studied. Previous studies have suggested an association between arsenic exposure and triglycerides, high-density lipoprotein cholesterol and the risk of hypertension [14]. However, most of the epidemiologic studies carried out previously have been cross-sectional and conducted in adults [15]. In this sense, in cross-sectional studies, urinary arsenic levels were positively associated with prevalent diabetes in populations from Canada (urine arsenic geometric mean 12.9 µg/L) [16]. However, in Bangladesh, the concentrations of well water arsenic were no associated with the prevalence of diabetes mellitus [17]. Despite a growing body of evidence on the role of low or moderate arsenic exposure and obesity [18-21], the association has remained unclear and some of studies that have previously examined these associations have thrown contradictory results. For this reason, we realized the present study with the aim

to evaluate the prospective association of prenatal exposure to arsenic and Body Mass Index (BMI) trajectories in a cohort of Mexican children's participants of a Prenatal Omega-3 Fatty Acid Supplementation and Child Growth Development (POSGRAD) birth cohort study.

Material and Methods

Study design and study population

In this study, we included a subsample of 450 pairs (mother-child), all participants of the POSGRAD birth cohort study aimed to evaluate the effect of DHA supplementation during pregnancy on the offspring's growth and cognitive development. A detailed description of the design and methods has been published previously [22]. Briefly, eligible women were 18 to 35 years of age with 18 to 22 weeks of gestation, and the intention to give birth in the General Hospital of Mexican Institute of Social Security (IMSS) in Cuernavaca (Morelos, Mexico). The subsample considered for the present report included 450 mother-child pairs with information about arsenic exposure measurements, as well as anthropometric variables. Information on sociodemographic characteristics and other maternal characteristics (obstetric history and lifestyle habits) were collected through interview-based questionnaires. Anthropometric measures were taken, and the Body Mass Index (BMI) was calculated from birth until 10 years or life with the height and weight data. All women signed an informed consent and the Emory University Institutional Review Board, the Committees of Biosafety, Investigation and Ethics from the National Institute of Public Health (INSP) and the IMSS General Hospital's Human Subjects Board approved this study.

Anthropometric measures of the child

Birth weight (to the nearest 10 g) and length (to the nearest 1 mm) were measured with the use of a pediatric scale and a portable length measurement board following standard procedures. Children's weight, height and Waist Circumference (WC) were measured by standardized procedures at 1, 3, 6, 9, 12, 18, 24, 36, 48, 60, 72, 84, 96, 108 and 120 months of life, with the same equipment and procedures [23,24]. Waist Circumference (WC) was measured using a Gulick fiber glasstape (Creative Health Products, Inc.) accurate to within 0.1 cm. Trained study personnel at the Mexican Social Security Institute's Hospital General I in Cuernavaca, Mexico, performed data collection. BMI was calculated with the height and weight data by the reference standards of the World Health Organization [23,24].

Exposure assessment to arsenic during pregnancy

First morning urine samples were collected from the mother between 18-22 weeks of pregnancy, containers were provided previously, and they were instructed on sample collection

procedures. The urine samples were refrigerated at 4°C and were then aliquoted and frozen at -70°C until the time of analysis. Arsenic concentrations were measured using inductively Coupled Plasma Optical Emission Spectrometry (ICP-OES) (Thermo Scientific) after a sample digestion step in the laboratory of the Technological Institute of Sonora. Briefly, 3 ml of the urine samples were placed in a linear microwave digestion vessel, 3 ml of concentrated nitric acid (HNO₃) and 1.5 ml of hydrogen peroxide (H₂O₂) were added, and the samples were digested (CEM Corp., Matthews, NC) for 35 min at 200°C. After microwave digestion, samples were adjusted to a final volume of 25 ml with water HPLC grade. For quality control purposes, blank and duplicate samples were analyzed during the procedure. SRM 3669 Standard Reference Material, Arsenic Species in Frozen Human Urine (NIST, Gaithersburg, MD) and CWW-TB Certified Wastewater Standard (High Purity, Charleston, SC) were analyzed to ensure assay quality. For arsenic, the recovery was between 82-106%, the coefficient of correlation was of 0.99 and the coefficient of variation was less than 8%. The detection limit of arsenic had an average value of 0.010 mg/L11.

Other maternal characteristics

At baseline, the participating women responded to a general health questionnaire that included information on weight and height before delivery, sociodemographic characteristics, health, and gynecological antecedents. During the 18 and 22 weeks of pregnancy, we administered an environmental questionnaire inquiring on water consumption, source of food and water, tobacco exposure, humidity, mildew, mold or saline formation on walls or floor, pet presence at home, presence of industry near home, and pesticide use, as well as 24-recall and FFQ dietary questionnaire. Data on age, sex, educational level of the mothers, and smoking status were collected via an interview. Height, weight, and waist circumference were collected by physical examination using a standardized protocol, and BMI was calculated by dividing the weight into kilograms by the square of height in meters.

Other information about the children

Information on age, sex, diet, health status during follows up, physical activity was obtained by questionnaire. As well as information about birth, breastfeeding and other environmental exposure. Details of the methods are included in the previous reports [11].

Statistical Analysis

First, baseline characteristics of the study population were performed using frequency measures and dispersion measures according to a variable type. For the exposure, the characteristics of the urinary concentrations of arsenic were described. Next, we

calculated the BMI for each subject using the measurements taken from birth to 10 years and the growth trajectories of the BMI for each child were evaluated. For the construction of the BMI growth curves, the age was used in months, from 0 (birth) to 120 months (10 years). A quadratic term for age was included to allow a non-linear BMI growth trajectory. Subsequently, a bivariate analysis was carried out, identifying the effect of exposure on growth trajectories at the different BMI measurement points; for this, a GEEs model was used. These effects were assessed considering the exposure variable in quartiles. For the model fit, an a priori approach was used to identify the main independent predictors of BMI (maternal age at the time of pregnancy, BMI before pregnancy, parity, type of delivery, passive smoking in home during pregnancy, schooling of the mother, socio-economic status). In addition, in the final model we included the interaction term between age in months and exposure in quartiles, the interaction term of the quadratic term of age in months and exposure. Finally, to evaluate the effect of the prenatal exposure to arsenic measured in quartiles on the BMI trajectories we use a General Equation Models (GEEs) for longitudinal data [25] adjusted by age in months, quadratic term for age in months, gender, weight before pregnancy, passive smoking in home during pregnancy and interaction term between exposure in quartiles and gender, age in months and quadratic term for age in months. For the exposure, the 1st. quantile was the reference category, and for sex, male was the reference category. Statistical significance was defined by an alpha level of 0.05. Analyses were performed with STATA software, version 14.0 (Stata Corp, College Station, TX).

Results

We included 450 mother-child pairs with complete information, all participants of the POSGRAD birth cohort study. Children included in the present analysis did not differ in terms of the main variables of the participants from the POSGRAD cohort sample and non-included in the analysis (data not shown). The mothers included in this study had a mean age of (+ SD) 26.1 (+4.8) at the second trimester. Socioeconomic status was categorized as low in 33.6%, medium in 36.4% and high in 30%. Considering BMI before pregnancy, about half of them had a normal BMI (54%), while 27.4 percent were overweight and 12.2% obese. 32.7% of these women were primipara. Only 2.1% of women reported smoking during pregnancy, however, 36.5% reported that someone in their home smoked during pregnancy. 48.6% of deliveries were normal and the rest caesarean, 53.6% of new-borns were male (Table 1). We did not observe significant differences between the participants included vs those not included for the main variables studied ($p > 0.05$) (Data not shown).

Table 1: Main characteristics of the study population.

Maternal characteristics	N	% or mean (SD)
Age at second trimester and enrolment (years)	450	26.1 (4.8)
Pre-pregnancy BMI (kg/m ²)	450	24.6 (4.6)
Underweight (<18.5)	28	6.1
Normal (>18.5–25)	245	54.3
Overweight (>25–30)	122	27.4
Obese (>30)	55	12.2
Parity	450	
Primiparous	148	32.7
Multiparous	302	67.3
Child sex	450	
Male	241	53.6
Female	209	46.4
Smoking during pregnancy	450	
Yes	9	2.1
No	441	97.9
Smoking someone's home	450	
Yes	164	36.5
No	286	63.5
Type of delivery	450	
Normal	218	48.6
Caesarean operation	232	51.4
Scholarship	450	
High school or more	252	56.1
Less than high school	198	43.9
DHA or placebo group	450	
DHA group	225	50
Placebo group	225	50
Child characteristics	N	($\bar{X} \pm SD$)
Birth weight (kg)	450	3.2 (2.9)
Length at birth (m)	450	0.54 (0.34)

WEIGHT (KG) AT 10 Y	450	29.3 (5.55)
HEIGHT (KG) AT 10 Y	450	137 (7.56)
Baz	450	0.04 (0.97)
BMI AT 10 YEARS OF AGE	450	20 (3.68)
Waist circumference (cm)	450	24 (11.76)

The mean and standard deviation of the concentration of arsenic during pregnancy is in Table 2. The mean concentration was 0.06 mg/L, SD (0.04 mg/L).

Table 2: Prenatal arsenic concentrations (mg/L) in urine samples.

Variable				
	Mean	SD	Percentile 25-75%	
*Arsenic	0.06	0.04	0.006	0.062
*Total arsenic concentration				

Regarding the metabolism indicators, 11.7% of the boys were above the 90th percentile for abdominal circumference (57.2 cm) and in girls; this figure was 5.3% (58.9 cm). 12.7% of the boys were overweight (BMI z-score > 1DE) (Data not shown).

We found a significant association between prenatal exposure to arsenic and the BMI trajectories from 0 to 10 years of age ($b_{\text{quartile2 vs. quartile1}} = 2.18$, ($b_{\text{quartile3 vs. quartile1}} = 2.54$, $p\text{-trend} < 0.05$) (Table 3, Figure 1). Finally, we observed an interaction effect between exposure and age in months, which indicates that the rate of change in BMI increases when the child's age increases, however, when the child is exposed to arsenic during pregnancy, the increase of the BMI is significant more accelerate in these population ($p < 0.05$) (Figure 2).

Table 3: Association between prenatal exposure to arsenic concentrations and BMI trajectories from 0 to 10 years of age in children's participants of the POSGRAD birth cohort study.

Variable	Arsenic exposure in quartiles					p-value of age in months and exposure interaction
	Quartiles	β	p-value	95% Conf. Interval		
*BMI trajectories	2 nd	2.18	0.032	0.211	4.173	0.008
	3 rd	2.54	0.011	0.613	4.526	0.001
	4 th	0.11	0.915	-2.125	1.902	0.837

GEEs models were adjusted by age in months, quadratic term for age in months, gender, weight before pregnancy, passive smoking in home during pregnancy and interaction term between exposure in quartiles and gender, age in months and quadratic term for age in months. Quartile 1 of exposure is the reference category. *Body Mass Index trajectories to 0 until 10 years of age.

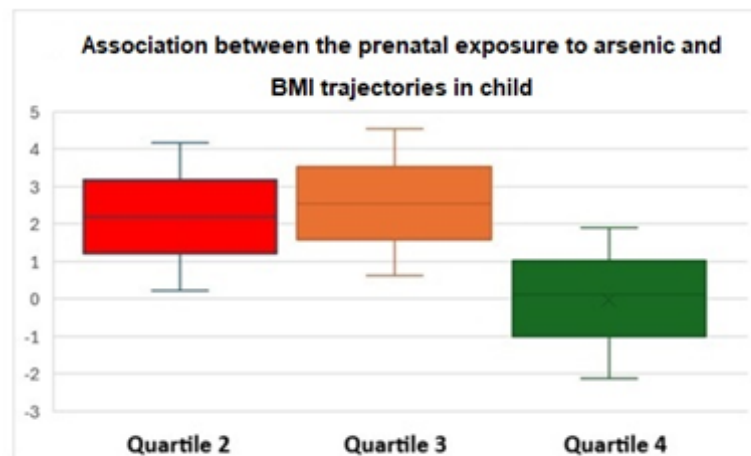


Figure 1: Association between BMI trajectories in children and prenatal exposure to arsenic.

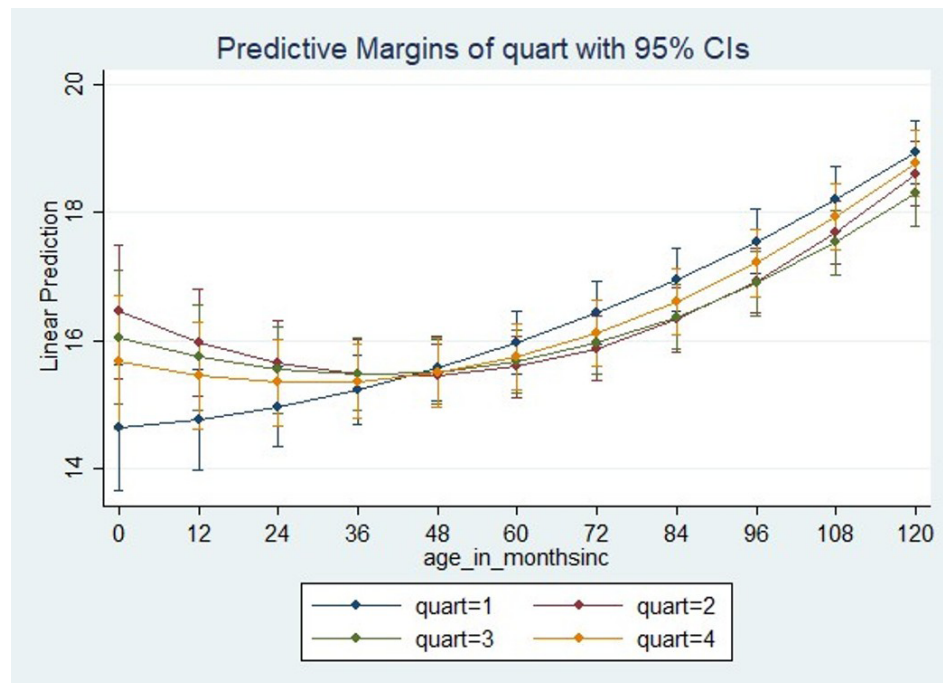


Figure 2: Predictive BMI trajectories in quartiles of arsenic concentrations with 95% CIs.

Discussion

The results of the present study strengthen the evidence that prenatal exposure to arsenic is associated with a faster rate of BMI gain through 10 years of age, which can be translated into higher risk of obesity especially if the BMI z-scores are already >0 at birth. This is a very important finding to public health, since this compound have the ability to interfere with the normal functioning of natural hormones [26] and may increase the risk of other important health problems (diabetes, hypertension and metabolic diseases) in early age of life. We found a positive association between prenatal exposure to arsenic and BMI trajectories. Our results are consistent with those reported in the literature, however, most of them are derived from experimental studies conducted in animals [27]. To our

knowledge, this is the first study conducted in Mexico with the aim to evaluate the relation between the prenatal exposure to arsenic and the longitudinal changes in BMI trajectories in children, using data from a birth cohort study, design that gives more support and strength our results. Xin Wang and collaborators utilized an Environmental Risk Score (ERS) that summarized the mixture of some heavy metals including the arsenic, found that an increase in ERS from 10th percentile to 90th percentile was significantly associated with 4.50 kg/m² (95% CI: 4.06, 4.94) higher BMI and 4.11 kg (95% CI: 0.83, 7.40) higher total body fat [28] but in the overall study population and not only in schoolchildren as in our study.

In a narrative review published in 2019, Farkhondeh T and colleagues reported that some epidemiological studies have shown that prenatal arsenic exposure can promote obesity, potentially through larger birth size and more rapid postnatal weight gain, elevated body fat content, and increased serum lipid profile, highlights that, the currently available evidence is insufficient to conclude that low-moderate dose arsenic is associated with obesity development [29]. Additionally, recent studies report that the possible biological mechanisms involved that can support our results are that inorganic arsenic (iAs) metabolism may disrupt metabolic function mainly. Experimental evidence indicates that some metals, such as arsenic (As), can act as an endocrine disruptor, affecting the functioning of hormonal receptors, specifically glucocorticoids, androgens, estrogens, and thyroids [30].

In addition, the toxicity of arsenic is influenced by its metabolism, after absorption, iAs is metabolized into mono- and di-methylated compounds (MMA and DMA) and the three arsenic forms are excreted in the urine, with DMA being more rapidly excreted via the kidneys. Lower methylation capacity is characterized by an increased MMA% compared with DMA% in urine and some investigators have indicated that high percentage of DMA in the participants' urine, reflecting the association between higher methylation capacity and increased risk of metabolic disorders [20]. In this sense, lower levels of MMA% and higher levels of DMA%, have been related to higher body mass index and diabetes-related outcomes in populations from Mexico, Taiwan, and the United States [12].

In summary, the proportions of inorganic arsenic, MMA, and DMA in an individual's urine can tell researchers something of the individual's risk for certain diseases: a high percentage of MMA (indicating lower methylation capacity) is a risk factor for cardiovascular disease, skin and bladder cancers, and the skin lesions that characterize highly exposed individuals, whereas a high percentage of DMA (indicating greater methylation capacity) has been associated with increased risk of diabetes.

On the other hand, some limitations must be considered when interpreting our results; first, the species of arsenic were not known, however, the lack of this information does not confuse the association found, but on the contrary, we think that it could be underestimated. Second, to have a single measurement of the exposure during pregnancy, limit the possible variations that could occur in the concentrations of the substances over the total period, however, we measured the concentration of arsenic at four years of age in children and the correlation found was correlated with the prenatal measurement, so we can assume that the contamination is continuous and we can assume that, the arsenic concentrations had the same behavior throughout the entire time of pregnancy. Third, the possibility of the selection bias (derived for the subsample selected for the birth cohort study), however, when comparing characteristics of the participants of the present sample vs those who were not included, we did not find significant differences. Four, to discard the possibility of confounding, the adjustment variables included in the models were all those that could be related to the exposure and the outcome of interest for this study; therefore, it is unlikely that this bias affects the results.

In addition, it is important to highlight the strengths of our research. To our knowledge, this is the first study conducted in a middle-income country to analyze the relationship between prenatal exposure to arsenic and the BMI trajectories in children in a contemporary birth cohort that provides greater internal validity to our results. Also, we obtained data prospectively from birth to 10 years of life, which reduces a possible classification error, minimizes the temporary problems and finally our sample size and repeated measures analysis gives more power to our findings.

In conclusion, the present study strengthens the evidence that prenatal exposure to arsenic may be a risk factor for subsequent risk of excess adiposity later in life. It is advisable to continue with the research, improving exposure assessment and studying the contaminants as mixtures should allow you to gain knowledge about the environmental determinants of growth and obesity and also because these markers may mediate/modify the risk of chronic disease later in life.

The data are available just for request to the corresponding author

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Author Contributions

ABV Project administration and write the paper; LHC participate in formal analysis; IAC collaborate in writing the paper; WP: supervision the paper; JOAM interpretation some results; IR Project administration

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Impact Statement

The prenatal exposure to endocrinal disruptor as arsenic may increase the risk of non-communicable diseases at early stage of age.

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