Environment International 121 (2018) 287–295

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Associations between school lunch consumption and urinary phthalate metabolite concentrations in US children and adolescents: Results from NHANES 2003–2014

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ARTICLE INFO

Handling Editor: Heather Stapleton

Keywords:
Phthalates
Diet
Cafeteria
Exposure assessment
Plasticizers
Endocrine disruptors

ABSTRACT

Diet is a major route of phthalate exposure in humans due to use in food packaging materials. School lunches may be an important contributor to phthalate exposure in children and adolescents in the US because of the large amount of packaging necessary for mass-produced foods. We used 2003–2014 National Health and Nutrition Examination Survey data to study the association between school lunch consumption and urinary phthalate metabolite concentrations in children (ages 6–11 years, N = 2196) and adolescents (ages 12–19 years, N = 2314). After adjustment for other covariates, children who Always consumed school lunch had significantly elevated urinary concentrations of the following phthalate metabolites compared to levels in children who Never ate school lunch: sum of di(2-ethylhexyl) phthalate metabolites, (28% higher, 95% confidence interval, CI: 10, 49%); mono-(carboxy-octyl) phthalate (MCOP; 43% higher, 95% CI: 17, 76%) and mono-*n*-butyl phthalate (18% higher, 95% CI: 3.5, 34%). We did not find statistically significant associations in adolescents, but the trend for MCOP concentrations was similar to that of children. In sensitivity analyses, associations between 24-hour recall of cafeteria food and urinary phthalate metabolites were not statistically significant, which could indicate that associations observed with Always consuming school lunch are due to residual confounding. Our findings show that children who Always eat school lunch had higher levels of exposure to some phthalates, but the source of differences in exposure need to be evaluated in additional studies.

1. Introduction

Phthalates are chemical compounds made from alcohols and phthalic anhydride, and are commonly used as plasticizers in a wide range of industrialized and consumer goods. Common applications of phthalates are personal care and household products as well as food packaging materials (Hauser and Calafat, 2005). Evidence suggests that phthalates are endocrine disruptors, with effects on androgenic (Gray et al., 2006) and thyroidal activity (Huang et al., 2018; Huang et al., 2007). Exposure during childhood has been associated with abnormal pubertal development (Meeker and Ferguson, 2014; Wolff et al., 2014; Wen et al., 2017), asthma and allergy (Hoppin et al., 2013; Bertelsen et al., 2013), increased blood pressure (Trasande and Attina, 2015; Trasande et al., 2013a), and perturbations in thyroid hormone levels (Weng et al., 2017). In addition, there is concern over phthalate exposure in childhood because of higher observed urinary metabolite concentrations in this age group compared to adults (Zota et al., 2014). The reasons for these differences are not fully understood.

Due to their common use in food processing and packaging materials (Serrano et al., 2014), a major route of phthalate exposure to humans is ingestion (Cirillo et al., 2011; U.S. Environmental Protection Agency, 2013; Department of Health and Human Services, 2009). Phthalates typically found in food packaging materials include di-*n*-butyl phthalate (DnBP), di-isobutyl phthalate (DiBP), di-cyclohexyl phthalate (DCHP), di-2-ethylhexyl (DEHP), di-*n*-octyl phthalate (DnOP) and di-isononyl phthalate (DiNP) (Zota et al., 2014; Department of

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https://doi.org/10.1016/j.envint.2018.09.009
Received 10 April 2018; Received in revised form 4 September 2018; Accepted 6 September 2018

Health and Human Services, 2009). Phthalates can transfer from food packaging to food, which presents an opportunity for ingestion (Serrano et al., 2014). Given that many US children and adolescents consume lunch prepared at school, this may be an important source of phthalate exposure. Previously, a study of school lunches in Italy found that a high proportion of school lunch foods tested contained DEHP and other phthalates (Cirillo et al., 2011). However, this study did not investigate the relationship between this exposure source and biomarkers of intake. Addressing this knowledge gap may inform exposure reduction strategies for these vulnerable age groups.

In this study, we explored the association between eating lunch prepared at schools and urinary phthalate metabolites in US children and adolescents. To address this question, we utilized data from the 2003–2014 sampling cycles of the National Health and Nutrition Examination Survey (NHANES) on children, ages 6–11 years, and adolescents, ages 12–19 years.

2. Methods

2.1. Study participants

NHANES is a nationally representative cross-sectional survey of the health and nutrition of the US population conducted by the National Center for Health Statistics (NCHS). The survey involves questionnaires and a physical examination performed in a Mobile Examination Center (MEC) where urine is collected for assessment of exposure to environmental agents on a subset of participants. For the present analysis we used data from the 2003–2014 sampling cycles. Participants age 6–19 years, who attended kindergarten through high school, with information on urinary phthalate metabolite concentrations, and who responded to the questionnaire on school lunch consumption were included in this analysis (total of 4510 participants). Response rates for participation in the examination component within this age range were good (75–85%) (Centers for Disease Control and Prevention, 2009a).

2.2. School lunch consumption assessment

School lunch consumption was identified from the questionnaire portion of the NHANES examination. Participants were asked, “During the school year, about how many times a week do you usually get a complete school lunch?” in the Diet Behavior and Nutrition section. This question was restricted to complete lunches provided by the school (Centers for Disease Control and Prevention, 2013). Answers were provided as a range of values, 0–5 days per week (Centers for Disease Control and Prevention, 2015a). The question was answered directly by participants, or by proxies (e.g., parents or caregivers) for participants younger than 16 years who could not answer it themselves (Centers for Disease Control and Prevention, 2015a). For our analysis we categorized the lunch consumption based on the value reported as follows: 0 days was categorized as Never; 1–4 days was categorized as Sometimes; and 5 days was categorized as Always.

In addition, we performed a secondary analysis using 24-hour dietary recall data to assess associations between energy intake from the school cafeteria and urinary phthalate metabolites. The purpose of this secondary analysis was to use different questionnaire information to ask the same research question, in order assess the validity of our findings. Dietary recall data is collected at MECs by questionnaire, and children ages 6–11 are aided in their responses by adults (Centers for Disease Control and Prevention, 2009b). We used variables reflecting total energy intake (kcal) over the past 24 h as well as total fat intake (% of total energy intake) over the past 24 h along with the food source variable to identify percentages of those values that came from school cafeteria food (Zota et al., 2016). We calculated associations between urinary phthalate metabolites and total energy intake from cafeteria food (measured in % kcal from cafeteria food) as well as total energy intake from fat that came from cafeteria food (measured in % kcal from fat in cafeteria food). Using the same approach as Zota et al. for examining associations between urinary phthalate metabolites and fast food consumption, we categorized these variables as Low (0% kcal from cafeteria food or from fat in cafeteria food), Medium (< median% kcal from cafeteria food or from fat in cafeteria food), and High (≥ median% kcal from cafeteria food or from fat in cafeteria food) (Zota et al., 2016).

2.3. Urinary phthalate metabolite measurements

Spot urine samples were collected from the subjects in MECs at the same time as the questionnaire information was collected. Samples were stored at temperatures below −20 °C prior to analysis by high performance liquid chromatography-electrospray ionization-tandem mass spectrometry (Centers for Disease Control and Prevention, 2010). The phthalate metabolites measured included: mono-(2-ethylhexyl) phthalate (MEHP), mono-(2-ethyl5-oxohexyl) phthalate (MEOHP), mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP), mono-(carboxynonyl) phthalate (MCNP), mono-(carboxy-octyl) phthalate (MCOP), mono-benzyl phthalate (MBzP), mono-*n*-butyl phthalate (MnBP), mono-isobutyl phthalate (MiBP), and mono-ethyl phthalate (MEP). Results were expressed in ng/mL. Most of the phthalate metabolites of interest were measured in the sampling cycles (2003–2014); however, MCNP and MCOP were measured beginning in the 2005–2006 sampling cycle. The description of the phthalate metabolite analysis conducted by the laboratory is described in detail elsewhere (Centers for Disease Control and Prevention, 2010). Phthalate metabolite concentrations below the limit of detection (LOD) were replaced with the LOD divided by the square root of 2 (Centers for Disease Control and Prevention, 2010).

In addition to examining individual phthalate metabolites, we calculated the molar sum of DEHP metabolites (ΣDEHP), expressed in nmol/mL, by the following formula in which each DEHP metabolite (ng/mL) is divided by the molecular weight of the chemical compound (g/mol) and then they are summed:

$$\sum \text{DEHP} = (\text{MEHP}/278.34) + (\text{MEOHP}/292.33) + (\text{MEHHP}/294.34) + (\text{MECPP}/308.33)$$

This measure is used to obtain an estimate of total exposure to the parent phthalate, DEHP (Barr et al., 2003). To examine distributions of phthalate metabolites, we corrected for urinary dilution by dividing the phthalate concentration by the urinary creatinine concentration to obtain final units of μg/g creatinine (μmol/g creatinine for ΣDEHP).

2.4. Statistical analysis

Analyses were conducted using R statistical software, version 3.4.0 (R Core Team, 2017). Because NHANES uses a complex sampling design to create nationally representative data, we used the package “survey” (https://cran.r-project.org/web/packages/survey/survey.pdf) to correct for oversampling of certain populations and to make the results generalizable to the US population. We constructed a new sampling weight for each participant per the NHANES Analytic Guidelines (Centers for Disease Control and Prevention, 2010).

First, we assessed characteristics of the study population, including age, body mass index (BMI) z-score, gender, race/ethnicity, family poverty income ratio (PIR), and sampling cycle, by school lunch consumption categories. Age was categorized into two groups including children (ages 6–11 years) and adolescents (ages 12–19 years). Age cutoffs were based on what is typically used to analyze NHANES data (Department of Health and Human Services, 2009). BMI z-score was calculated using World Health Organization (WHO) reference curves which incorporate information on BMI (kg/m^2) as well as age and gender (World Health Organization, 2017). BMI z-scores were then categorized as follows: < 1, “normal and underweight”; 1–2,