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Dietary Predictors of Urinary Environmental Biomarkers in young girls, BCERP, 2004–7

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Abstract

Background—Exposures of children to phthalates, parabens, and bisphenol-A (BPA) are of concern because of their hormonal potential. These agents are found in a wide range of foods and packaging. We investigated whether intake of certain foods predict exposures to these chemicals in young girls.

Methods—Among 1101 girls (6–8 years at enrollment) from the Breast Cancer and Environment Research Program (BCERP) study, we measured urinary exposure biomarkers for phthalates, parabens, and BPA and assessed dietary intake using 24-hour recall 2–4 times. We examined the average daily servings of major and minor food groups categorized as 0- <0.5, 0.5 – < 1 and ≥ 1 servings per day. Items included dairy, eggs, fats, fish, fruit, single grains, meat, non-poultry meats, pasta, poultry and vegetables. Covariate-adjusted least squares geometric means and 95%

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confidence intervals of creatinine-corrected phthalate and phenol metabolite concentrations in urine were calculated in relation to food intake.

Results—Grains, flour and dry mixes and total fish consumption were positively associated with BPA and the sum of four di-2-ethylhexylphthalate (DEHP) urinary metabolite concentrations. Non-fresh vegetables and poultry were both positively associated with BPA and paraben urinary concentrations. Fats, oils and poultry consumption were positively associated with BPA. Whole-fat dairy consumption was associated with ΣDEHP.

Conclusions—Some foods may contribute to child exposures to certain chemicals, and this may constitute modifiable means to reduce these environmental exposures.

Keywords

biomarkers; endocrine disruptors; phthalates; bisphenolA; parabens

1. Introduction

Phthalates, bisphenol A (BPA), and parabens are chemicals widespread in our environment, derived from many sources. Human exposure can occur through inhalation, ingestion, and dermal contact. Oral and inhalation pathways lead to much more efficient uptake of phthalates into the human body than the dermal pathways (Wormuth et al. 2006), making diet an important route of exposure (Lakind and Naiman 2011). Phthalates in plastic food packaging and canned food linings are not chemically bound to the products so they can be leached into foods (Cao et al. 2010; Kang and Kondo 2002). Measurable levels of both phthalates and BPA have been identified in a wide range of foods including baby food, canned beans and meat (Fromme et al. 2007; Schecter et al. 2013; Tsumura et al. 2001) and increased levels of their metabolites in humans have been associated with certain kinds of foods and packaging including poultry, canned vegetables, poultry, dairy and soda (Colacino et al. 2010; Schettler 2006) (Braun et al. 2011; Lakind and Naiman 2011; Trasande et al. 2013)

Phthalates are additives in many common consumer products such as food packaging, vinyl flooring, and personal care products (e.g., fragrances, cosmetics). BPA is used in the production of epoxy resins and polycarbonate plastics. BPA is found in canned and plastic food and drink packaging, dental sealants, adhesives and varnishes. Parabens have been used as preservatives in foods, drugs and cosmetics for over 50 years. They have effective antimicrobial activity and relatively low toxicity in humans (Darbre and Harvey 2008; Soni et al. 2001; Soni et al. 2002). Methyl paraben (MP) and propyl paraben (PP) are the two most common commercially-used parabens (Andersen et al. 2010; Soni et al. 2001; Soni et al. 2002).

Phthalates, BPA, and parabens are all considered endocrine disruptors (EDs); such compounds can mimic endogenous hormones, antagonize hormone function, alter the synthesis and metabolism of natural hormone or modify hormone receptors (Diamanti-Kandarakis et al. 2009). Exposure to EDs has been shown to adversely affect a range of human health endpoints, including reproductive function and thyroid activity in both males

and females (Barlow and Foster 2003; Hauser et al. 2006; Latini et al. 2003; Meeker et al. 2011).

Measurable concentrations of phthalate metabolites, BPA and parabens are detected in the urine of over 90% of the U.S. population (CDC 2009). Most phthalates and BPA concentrations are normally higher in children and minority groups (Calafat et al. 2010), while DEP and paraben concentrations are generally higher in adults. We examined whether urinary biomarker levels were associated with increased intake of certain types of foods in a population of young girls. Furthermore, previous research has shown increased chemical exposure in humans associated with food packaging (Mariscal-Arcas et al. 2009; Vandenberg et al. 2007). Therefore, we also examined whether chemical exposure differed by specific types of food packaging and preparation. Identification of food sources will provide a way to reduce exposure to these chemicals through avoidance of these foods.

2. Materials and Methods

The Breast Cancer and Environment Research Program (BCERP) study population of girls 6–8 years at enrollment (2004–2007) in three U.S. sites, New York City, Cincinnati, and San Francisco Bay Area, has been previously described (Biro et al. 2010). Briefly, Mount Sinai School of Medicine (NYC), recruited through clinics, schools, and neighborhood centers in East Harlem, New York; Cincinnati Children's Hospital/University of Cincinnati (Cincinnati), recruited through schools in the Cincinnati metropolitan area and through the Breast Cancer Registry of Greater Cincinnati; and the San Francisco Bay Area group recruited Kaiser Permanente Northern California Health Plan members in the San Francisco Bay Area. For this analysis, 1101 girls with baseline urinary biomarker measurements, diet, anthropometric and questionnaire data were included.

2.1 Dietary Data

Twenty-four hour dietary recalls for all participants were performed at the Cincinnati Center for Nutritional Research and Analysis using the Nutrition Data System for Research (NDSR). Regarding usual intake, there is evidence that the detailed information obtained from 24 hour diet recalls may provide more accurate estimated than information from FFQ's (Schatzkin et al. 2003). Dietary recalls were conducted with participant caregivers over the telephone every 3 months during the first year after enrollment. Girls with two - four 24-hour dietary recalls collected during the year after baseline interview were included; values were averaged to estimate daily (24 hour) intake in grams. This was converted to servings which is the unit of analysis hereafter. Dietary recalls where total kilocalories were <400 or >4000 were considered outliers and excluded (n=18) (Willett 1998). Similarly, diet recalls that were completed more than a year after the baseline urine sample were excluded (n=23). Remaining girls after these exclusions were included in this analysis (n=1101).

Food groups were based on the USDA MyPyramid equivalents (United States Department of Agriculture 2010) and NDSR food groupings (Table 1). We calculated the average daily servings of the following food groups: dairy (including low and whole fat), eggs, fats (including vegetable oils, olive oil, butter, lard), fish, fruit, meat, (beef, lamb, pork, game), poultry, and vegetables (including legumes, and vegetable juices) and total grains. Grains

consist of many different types of foods, therefore grains were examined as NDSR assigned subgroups rather than as a single combined group (Table 2).

During the dietary recall process, participants describe details about the food preparation. NDSR translates this information into a detailed list of ingredients. We used this list to develop food sub-categories that reflect sources of exposure from food processing and packaging by scanning records (n=4500) for key words that differentiate non-fresh from fresh foods. Non-fresh foods were foods that had key words denoting commercial processing or packaging such as ‘canned’, ‘commercial’, ‘coated’, ‘dehydrated’, ‘fast food’, ‘flavored’, ‘frozen’ and ‘processed’. For example, we categorized kidney beans as fresh when described as ‘vegetables, beans, kidney beans, cooked from dried’ and as non-fresh when described as ‘vegetables, beans, kidney beans, canned –drained, regular’. In some instances where a fresh food was considered to be part of a processed food, it was categorized as non-fresh. For example, the potato component in French fries is described as “vegetables, potato, without skin.” It was necessary to look at the whole food description to correctly categorize as non-fresh. The categorization of fresh and non-fresh foods was done predominantly for vegetables (which included legumes) and fruits, as these foods are available for purchase either fresh or with packaging or processing, whereas foods like meats, dairy, and grain-based food are generally packaged or processed. Additionally we broke down the NDSR variable named ‘Grains, flour, and dry mixes’ as this grouping contained grains which are part of other foods (flour from mixed foods). Utilizing these approaches, we created food sub-groups (Tables 2 and 3) which were used as food predictors in our analyses.

Foods were categorized as 0- <0.5, 0.5 – < 1 and ≥ 1 servings per day (low, medium and high intake) based on reported intake. Our mean daily servings are lower than USDA recommendations; although the highest category (≥ 1 servings per day) approximates the USDA age recommended total daily amounts for several of the food groups. To calculate the proportion that a subgroup contributes to the total food group, as reported in Results and figures, we first calculated the total number of servings/day of a food group for all girls, for example 100.91 servings /day of fish among the 1101 girls. Then we divided the total subgroup servings (for ex. 12.9 servings of fried fish) by the total in the food group (100.91) to obtain the proportion for a subgroup (12.9%). We categorized meal locations into 3 groups: home; school; and restaurant/deli.

To supplement the data collected through the 24 hour dietary recalls, we ascertained additional sources of dietary exposure in the baseline questionnaire. Girls were asked the weekly frequency of consumption of canned beverages and foods, as well as meats or cheeses that came in plastic wrap or containers during the week and month prior to the interview and urine collection. We examined the exposures derived from these questions separately from the 24 hour recall data.

2.2 Urinary biomarker measurements

Baseline urine samples were analyzed at the National Center for Environmental Health at the CDC. Laboratory analytic methods have been published (Kato et al. 2005; Ye et al. 2005; Ye et al. 2006). Urinary conjugates of the target analytes are enzymatically

hydrolyzed, concentrated and separated from other urine components by on-line solid phase extraction coupled to high performance liquid chromatography. Quantitation is achieved by isotope dilution tandem mass spectrometry. Limits of detection (LODs) were calculated as three times the standard deviation of near-zero or blank quality control specimens. Analytic quality control and reagent blank samples included in each analytical batch met the CDC reporting guidelines. All specimen collection and storage materials were supplied by the CDC. The CDC laboratory is certified by the Health Care Financing Administration to comply with the requirements set forth in the Clinical Laboratory Improvement Act of 1988. In addition, QC samples from an external pool were incorporated at each site before shipping. As reported, results of these masked specimens were consistent between sites and batches (Wolff et al. 2010). For the 13 phthalate and phenol metabolites included here, the CV's were less than 10% for 8 biomarkers and 10–20% for 5 analytes (n=101 control pool specimens). Urinary concentrations were obtained for creatinine and nine phthalate metabolites: monoethyl phthalate (MEP), mono-n-butyl phthalate (MBP), mono-(3-carboxypropyl) phthalate (MCPP), monobenzyl phthalate (MBzP), mono-isobutyl phthalate (MiBP), mono-(2-ethylhexyl) phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP), and mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP); four phenols (BPA, n = 1,149; methyl-, butyl-, and propylparabens, n = 1,059). Butyl paraben and MEHP had the highest %<LOD (49% and 19%, respectively). We used log-transformed values of the urinary biomarker concentrations to normalize the distributions, and we substituted the value LOD/✓2 for concentrations below the LOD for the statistical analyses. To reduce multiple comparisons and to examine phthalates previously associated with food sources, we combined the phthalate metabolites into three groups based on molar sums that represent similar sources and similar biologic activity; low molecular-weight phthalate metabolites (low MWP: MEP, MBP, and MiBP), high molecular-weight phthalate metabolites (high MWP: MCPP, MECPP, MEHHP, MEOHP, MEHP and MBzP) and the sum of four DEHP metabolites (∑DEHP: MEHP, MEHHP, MEOHP, MECPP). Similarly, a molar sum of methyl and propyl parabens was created (∑paraben) expressed as propyl paraben (molecular weight 180.2); butyl paraben was excluded because of the high number of <LOD observations. Concentrations are reported corrected for urinary creatinine (μg/g creatinine) to account for urine dilution. The results for ∑DEHP and high MWP were very similar; therefore only the results from ∑DEHP analyses are presented for ease of comparison with other studies.

2.3 Statistical Analysis

Covariate-adjusted least squares geometric means and 95% confidence intervals of creatinine-corrected phthalate and phenol metabolite concentrations in urine in relation to food intake were calculated using PROC GLM in SAS 9.2. Potential confounders were considered as covariates if the bivariate analyses showed differences in both diet and biomarker distributions by confounder category ($p < 0.05$ pearson's Chi-square). Caregiver's education (categorized as high school diploma or less and some college or more) was used as an indicator of socioeconomic status and met the above criteria. Diet and biomarker levels also differed by child's race/ethnicity and age at urine collection, so these were also included in the final models. Total calories did not meet the definition of true confounder in our data and body mass index percentile (BMI%) was not included as it is a

potential collider in causal pathway models (Hernan and Cole 2009). As stated above, the biomarker concentrations are creatinine corrected to reduce misclassification due to different urine dilutions. There were differences in mean servings of certain foods and levels of biomarkers by site, and it is possible that specific food sources or processing differed by site, so we reran the models including site. Models with and without site provided almost identical results. Since we had no specific evidence to show that food sources of these chemicals differed by geographic region, we excluded site as a confounder. Therefore, only results from models without site adjustment are presented, which also allowed us to retain range of diet exposure gained by this multi-site study.

We examined the association between meal location and urinary biomarkers (without foods), adjusted for total meal count per girl. We also examined meal location as a possible covariate in the final model.

2.4 Final Model

Geometric means of urinary biomarkers were adjusted for age, caregiver education, and race/ethnicity. A trend test identified linear associations between food servings groups and urinary metabolites. These were obtained using the median food servings per food category as a continuous variable in the model. Two-sided P value for trend < 0.05 was considered to be statistically significant. We separately examined dietary intake from only the diet call closest to the urine collection, and results were similar to intake from the average of the calls over a year.

3. Results

The population has been described elsewhere (Biro et al. 2010). Girls were 6–8 years old at the time of urine collection; there were similar proportions of White, Black and Hispanic girls. Several baseline characteristics differed by site, as previously reported. Dietary intakes differed by important characteristics including site, BMI% and race (Table 1). There were small differences in diet patterns among the sites, with California girls reporting higher servings of vegetables, grains and fats and the least amount of meat. Cincinnati girls had a higher proportion of canned foods. The mean servings of major food groups were comparable to national data for girls in this age group except for fruit, dairy and grains (Table 1). For most food groups, our data as well as the national means, do not meet the USDA serving recommendations. We calculated means servings for several food subgroups we analyzed (Table 2). In terms of meal location: 78% of the girls' meals were eaten at home, 11% at school and 7% at either a restaurant or deli (data not shown).

Several food groups were associated with increased urinary concentrations of BPA and ΣDEHP. Increased "grains, flours and dry mixes" (which include rice, oatmeal and flour in mixed foods) and increased total fish consumption were positively associated with BPA and ΣDEHP urinary concentrations. Review of specific types of grains included in this broad category, revealed that "flour in mixed foods" had a strong association with BPA and that rice was associated with ΣDEHP. Rice contributed approximately 35% of grain consumption, and "flour in mixed foods" contributed over 42% of grain consumption. The sub-category, "fried, fast food type fish", made up 30% of the total fish consumption

category and was significantly associated with both BPA and ΣDEHP. Among girls with higher fish consumption (>= 1 serving/day), 18% of the servings were fried, processed/fast food (adjusted geometric mean ΣDEHP 267 μg/gC) compared to only 2.7% of the servings among girls with lower fish consumption (adjusted geometric mean ΣDEHP: 165 μg/gC). Canned fish accounted for 30% of all fish consumption, but when we examined canned fish alone we did not see any associations. Greater total vegetable intake was associated with increased BPA concentration (Table 3). To further examine the relationship between vegetable intake and BPA, we divided the total vegetable servings into two groups; non-fresh (53%) and fresh (47%). Figure 1 shows the items that make up non-fresh vegetables. Non-fresh vegetables were positively associated with BPA and paraben urinary concentrations. We did not see similar associations for non-fresh fruit (44% of total fruit intake). However, we found that fresh fruit was inversely associated with MBzP, a high MWP metabolite, but not with high MWP or ΣDEHP. Figure 2 shows the types of foods that make up canned foods, the majority being tomato-based pasta and pizza sauces, and canned or frozen beans or corn.

Fats and oils, which include margarine, oils, shortening, butter and animal fats, were positively associated with BPA. Whole-fat dairy consumption was associated with ΣDEHP. Greater poultry consumption was positively associated with BPA and Σparabens and low MWP (Table 3). Poultry includes fried and processed chicken. The association for low MWP was seen in the total poultry and fried/processed chicken category whereas for BPA and Σparabens, the association was seen for “total chicken”.

No associations were found when we examined meal location alone in relation to biomarkers. When we included the variable meal location in the final model as a covariate (including food variables), associations were similar to the models without it.

We did not find associations of biomarkers with reported intake of canned food and beverages or in plastic packaging, consumed in the prior week and month of the interview, as reported in the baseline questionnaire.

4. Discussion

We found that certain foods predicted higher urinary concentrations of BPA, parabens and ΣDEHP. Our research did not identify exact sources of contamination nor when it may have occurred in the food chain, but the associations we observed are consistent with reports in the literature that link these chemicals to food processing and packaging, and possibly to indirect sources such as cattle feed (Jarosova A 2010). Association of BPA with non-fresh vegetables is reported by others (Braun et al. 2011), including detection of BPA in canned foods (Cao et al. 2010; Kang and Kondo 2002; Schecter et al. 2010). A recent risk assessment suggests that canned vegetables and canned fruits have different contributions to total BPA; canned vegetables contribute 10–40% of the daily BPA intake, whereas canned fruits contribute 3–6% (von et al. 2010). Our results for these two food groups are in line with this and other studies (Braun et al. 2011). Noonan et al. (2011) showed that BPA concentrations in canned foods vary greatly not only between food types but also within food types and between production lots from the same manufacturer. Several factors

influence the amount of migration of BPA into canned goods, including the matrix, heat during the sterilization process and the specific kind of can coating. However the extent that these factors determine migration into cans has not been quantified (Grumetto et al. 2008). Parabens in our population were associated with poultry; however they were not related to some foods such as sugary items, marmalades, jellies, baked goods and processed fruits, as had been reported by others (Saad et al. 2005; Soni et al. 2002).

We found relationships between grains, dairy and fish and ΣDEHP, whereas others found associations between poultry or vegetables and ΣDEHP using NHANES data (Colacino et al. 2010). They also found associations with low MWPs and fruits, vegetables and meats. We did not see the latter possibly because our cohort is not the same as NHANES with regards to race, age and geographic location. Additional reasons for differing results could be study design, different dietary assessments, serving cutpoints, changes in packaging over time and foods not being a substantial source of LMW phthalates. Colacino's study was cross-sectional using one recall the day before urine was collected whereas our dietary data were collected 1–12 months after urine collection. We used dietary servings in 3 categories for each food group whereas Colacino et al. used dietary servings as a continuous variable. Dietary intake measurements attempt to preserve relative rankings rather than to provide accurate continuous variables (Willett 1998). Therefore use of categories (low, medium and high) of food consumption is a more conservative approach than a linear model.

Our association between whole fat dairy consumption and ΣDEHP is supported by studies that have reported similar relationships and that have detected phthalates in dairy products (Colacino et al. 2010; Petersen and Jensen 2010; Sharman et al. 1994). Aluminum paper-foil laminate of butter packaging and dairy tubing are cited as possible sources of DEHP (Sharman et al. 1994). Other foods have also been found to be related to higher DEHP metabolites concentrations, including grains, poultry and fish (Colacino et al. 2010; Fromme et al. 2007a; Lakind and Naiman 2011; Schecter et al. 2013). Two studies that performed quantitative comparison of dietary intake based on duplicate samples with imputed intake from excreted DEHP showed that food is the dominant intake source of DEHP (Fromme et al. 2007; Wormuth et al. 2006) and important sources of dibutyl phthalate and BBP (Wormuth et al. 2006). In contrast, diet accounts for a small fraction of exposure to low MWPs (dimethyl phthalate and diethyl phthalate) which are predominantly used in personal products, such as shampoo (Wormuth et al. 2006). Nevertheless, we and others find associations of low MWPs with certain foods, such as poultry and vegetables (Colacino et al. 2010) and decreased after a fresh food intervention (Rudel et al. 2011).

A number of other studies report food packaging and BPA associations, including carry-out food wrapping (Vandenberg et al. 2007), cans and microwave containers (Carwile et al. 2009; Mariscal-Arcas et al. 2009), and canned tuna fish (Munguia-Lopez et al. 2005). Packaging is thought to be the primary source of phthalate contamination of foods as well. An intervention study in families showed BPA and DEHP exposures substantially reduced when diets were restricted to foods with limited packaging (Rudel et al. 2011). We did not see any associations of urinary biomarkers related to consumption of foods likely to come into contact with these types of packaging when using our self-report questionnaire data. Retrospective and concurrent self-report questions of packaging and canned consumption

have the potential to be imprecise from inaccuracies in memory or estimation, which could lead to non-differential misclassification, biasing the results towards the null. Recency has been found to influence children's recall accuracy; a study found that shortening the retention interval of dietary recalls increases accuracy for reporting energy and macronutrients (Baxter et al. 2004).

Our results suggest that foods like grains and poultry increase urinary concentrations of metabolites of the high and low MWPs, while meat consumption does not. The fact that we don't see an association with meat is somewhat surprising since parent phthalate diesters are lipophilic and are released from packaging mainly into foods containing fat, such as meats and chicken but not grains. Therefore, there may be alternative routes of food contamination by phthalate diesters for chicken and grains. Colacino also saw differences in associations for chicken and meat (Colacino et al. 2010). Reports show that agricultural crops and animals have contact with these chemicals during cultivation, and such activities probably differ for chicken from meat-producing animals. The NDSR "grains, flour and dry mixes" category includes foods that are not exclusively composed of grains. For example, flour used in pizza is attributed to this category, however pizza also contains other ingredients such as tomato sauce that has more opportunities for contamination during processing and packaging. Pizza's tomato sauce would be assigned to another food group. This highlights the complexity of the NDSR processing of reported foods and in terms of the data structure, we cannot disentangle exposure sources from each of the ingredients in the food. It was somewhat unexpected that we did not find associations with grain specific groups, such as cookies, pastries and breads. Chemical analysis of foods before and after they reach the market shelves would be one step to understand the points of contamination of our food supply. Colacino et al. suggest consultation from the food industry to help determine what step in the production process contamination is occurring (Colacino et al. 2010).

Our null results when examining meal location differ from Lakind et al. who finds a positive association between BPA and meals not prepared at home and school lunches per week in a population sample (NHANES). One main difference is that our meal location variable does not specify where the meal was prepared, so when a child reports eating at school, we can not differentiate whether it is a school lunch or a meal prepared at home.

There are several limitations to this study. Our study population was recruited from certain groups within 3 geographic locations with most girls residing in either a city or suburban location. Therefore our results are not representative of all US girls. The majority of the girls were born in the United States; however some of their parents were not. Although different dietary habits exist, including purchasing and cooking between first and second generation (Liu et al. 2012) we did not have the data available to analyze this. We used only one sample to characterize each girl's urinary biomarker level. Because phenol and phthalate metabolites are relatively shortlived in the body, a single sample may not represent the temporal window of exposure relevant to outcomes. An increasing number of reports now document intraindividual variability of urinary phenols and phthalate metabolites over time, among pregnant women (Braun et al. 2011) and adults, including men and women (Fromme et al. 2007; Hauser et al. 2004; Hoppin et al. 2002; Peck et al. 2010), and families including children (Ackerman et al. 2014). Before undertaking this research we conducted a study to

establish the reliability of such measurements in children and found that intraindividual variability in children was acceptable over about 12 months (Teitelbaum et al. 2008). Study designs report varied intervals between urine collections, ranging from days to years; studies collect spot samples at various times of day, and they differ with regard to age and sex of subjects. ICC's (intraclass correlation coefficients) suggest poor to good reliability of a single urine measurement for phthalates (fair-good; ICC ~0.2–0.8), parabens (fair; ICC 0.3–0.6; Meeker; Smith), and BPA (poor; ICC 0.1–0.2) in various reports. However, several studies indicate that ranked exposure categories (e.g. tertiles of urinary biomarkers) are quite consistent over time even when the ICCs are poor (Hauser et al. 2004; Peck et al. 2010). Some phthalate metabolites show better agreement over time (Fromme et al. 2007; Hauser et al. 2004; Hoppin et al. 2002) than others (Ackerman et al. 2014; Fromme et al. 2007; Peck et al. 2010; Preau, Jr. et al. 2010). In the case of this paper, dietary data were collected 2–18 months after the urine specimen. As a further check on temporality of urine measures and diet in this paper, when we limited the dietary data to the call closest to the urine collection, the findings were not improved. The dietary intake method used is primarily designed to gather nutritional information, not to specifically provide the differentiation between fresh and non-fresh foods (processed and packaged). This limited our ability to identify sources of exposure for certain food groups. Lastly, diet alone cannot explain the total source of exposure as there are additional sources of phthalates, BPA and parabens, such as air pollution, personal care products, floor coverings in housing and medications, and these were not considered. We have obtained a detailed inventory of the girls' exposure to many of these sources and this analysis is the subject of future publication. Together with the dietary data, this information will give us a more complete picture of the total body burden of these chemicals.

Our study has several strengths. Dietary data were the average of 2–4 calls within a year, which has shown to be a reliable measure of usual dietary patterns, including seasonal variation. The very detailed NDSR information allowed us to create a more thorough description of food source (e.g., canned, processed, fresh). Finally, our sample size is relatively large and comprises a diverse group of girls with variable exposures and food habits.

EDs interfere with hormone action, potentially affecting a wide range of health effects, and food is a significant contributor to human ED exposure. Better information on identifying when and how contamination occurs in the food chain may enable children to maintain a healthy diet with less concern about adverse exposure.

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Highlights

- BPA and phthalates are found in a wide range of foods and packaging.
- Consistent with others, whole fat dairy products were associated ΣDEHP
- Grains, fish, non-fresh vegetables, poultry and fats were associated with BPA.
- Grains, fish were also associated with ΣDEHP concentrations.
- Foods contribute to children's exposures to certain chemicals

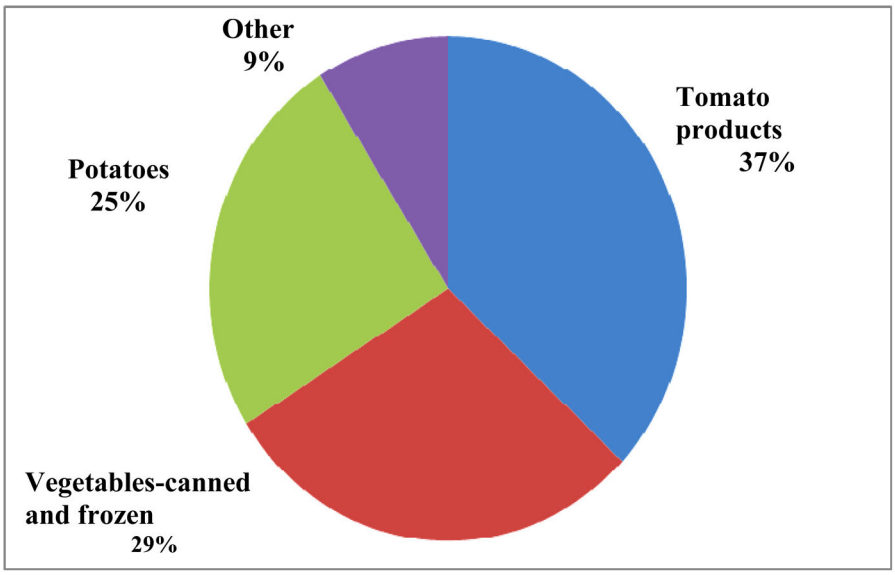

Figure 1.

Specific foods and food groups that make up non-fresh vegetable consumption in BCERP cohort 2004–7. Potatoes include french fries, mashed from dehydrated, hash browns and tater tots. Canned and frozen vegetables include: peas, corn, frozen peas, spinach and collards. Tomato products include sauces and pastes from jars and cans. “Other” includes mainly canned beans.

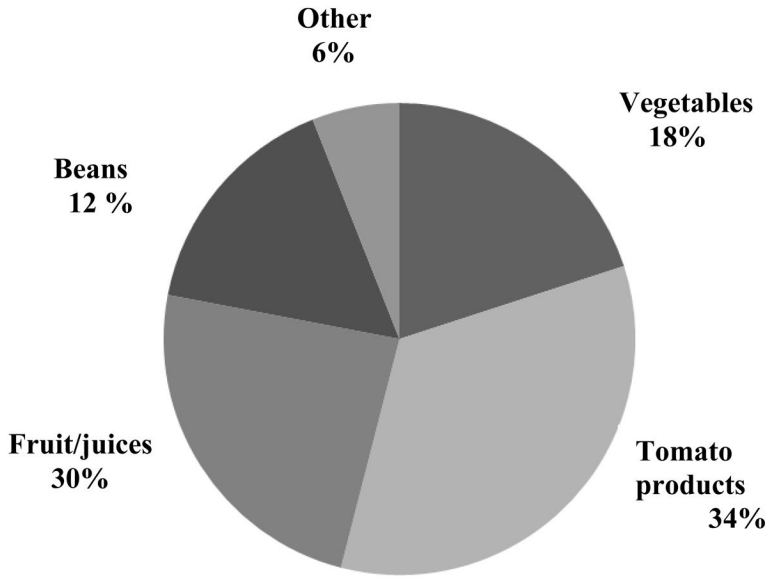

Figure 2.

Specific foods that make up canned good consumption in BCERP cohort 2004–7. “Other” includes fish, meats and gravies.

Table 1

Mean (standard deviation, std) servings per day of major food groups measured at baseline examination year according to personal characteristics in 1101 girls[a]: BCERP cohort 2004–2007.

			Grains	Dairy	Vegetables	Fruit & fruit juice	Meat including poultry	Fish	Eggs	Fats & oils	Total canned food
		N	Mean (std)	Mean (std)	Mean (std)	Mean (std)	Mean (std)	Mean (std)	Mean (std)	Mean (std)	Mean (std)
Age at urine donation (year s)	6.0 – 6.9	277	5.42 (1.65)	1.74 (0.78)	1.41 (0.8)	2.05 (1.33)	2.64 (1.36)*	0.25 (0.52)	0.23 (0.33)	1.92 (1.3)	0.51 (0.45)
	7.0 – 7.9	583	5.60 (1.71)	1.79 (0.83)	1.48 (0.83)	2.14 (1.41)	2.60 (1.34)	0.29 (0.65)	0.24 (0.33)	2.04 (1.29)	0.49 (0.46)
	≥ 8	241	5.61 (1.68)	1.85 (0.88)	1.54 (0.91)	2.01 (1.32)	2.93 (1.47)	0.27 (0.63)	0.22 (0.29)	2.06 (1.27)	0.56 (0.49)
Race/ethnicity	White	383	5.97 (1.54)*	2.01 (0.88)*	1.52 (0.95)*	1.97 (1.36)*	2.20 (1.26)*	0.18 (0.43)*	0.18 (0.29)*	2.05 (1.29)*	0.50 (0.43)*
	Asian	56	5.68 (1.56)	1.69 (0.84)	1.52 (0.72)	1.87 (1.11)	2.53 (1.28)	0.51 (0.91)	0.28 (0.33)	2.09 (1.27)	0.40 (0.36)
	Hispanic	325	4.94 (1.79)	1.81 (0.78)	1.33 (0.77)	2.33 (1.35)	2.62 (1.29)	0.22 (0.47)	0.28 (0.35)	1.78 (1.39)	0.40 (0.43)
	Black	337	5.67 (1.60)	1.53 (0.74)	1.55 (0.77)	2.02 (1.42)	3.31 (1.37)	0.40 (0.80)	0.24 (0.33)	2.19 (1.15)	0.64 (0.51)
BMI- %ile at urine collection	< 50	371	5.69 (1.75)*	1.81 (0.90)*	1.41 (0.79)	2.01 (1.31)	2.53 (1.40)*	0.28 (0.63)	0.21 (0.30)	1.94 (1.28)*	0.48 (0.45)
	50 – <85	374	5.64 (1.74)	1.86 (0.80)	1.50 (0.90)	2.17 (1.32)	2.67 (1.35)	0.28 (0.63)	0.24 (0.33)	2.16 (1.36)	0.53 (0.46)
	≥ 85	355	5.33 (1.54)	1.70 (0.78)	1.52 (0.83)	2.10 (1.47)	2.85 (1.37)	0.27 (0.60)	0.25 (0.34)	1.95 (1.21)	0.52 (0.49)
Caregiver's education	≤ HS diploma	308	5.14 (1.84)*	1.78 (0.77)	1.32 (0.76)*	2.17 (1.38)	2.78 (1.36)	0.23 (0.56)	0.24 (0.32)	1.87 (1.38)*	0.43 (0.46)*
	≥Some college	766	5.73 (1.56)	1.79 (0.86)	1.54 (0.87)	2.06 (1.37)	2.62 (1.40)	0.30 (0.64)	0.23 (0.32)	2.08 (1.25)	0.54 (0.46)
Site	NY	351	4.96 (1.69)*	1.68 (0.78)*	1.34 (0.73)*	2.24 (1.45)*	2.96 (1.39)*	0.29 (0.60)	0.27 (0.34)*	1.91 (1.26)*	0.47 (0.48)*
	OH	329	5.75 (1.53)	1.95 (0.83)	1.39 (0.79)	1.67 (1.20)	2.79 (1.36)	0.22 (0.64)	0.16 (0.26)	1.93 (1.20)	0.62 (0.48)
	CA	421	5.91 (1.67)	1.75 (0.86)	1.66 (0.93)	2.29 (1.36)	2.37 (1.32)	0.31 (0.61)	0.26 (0.34)	2.17 (1.36)	0.45 (0.42)
All BCERP participants		1101	5.56 (1.69)	1.79 (0.83)	1.48 (0.84)	2.09 (1.37)	2.68 (1.38)	0.28 (0.62)	0.23 (0.32)	2.0 (1.30)	0.51 (0.46)
NHAN ES[1]		215	6.66 (3.87)	2.61 (2.87)	0.96 (0.47)	1.14 (1.49)	2.92 (1.97)	0.25 (0.72)	0.27 (0.49)	NC[2]	NC[2]

[a] Table includes all study participants with both dietary recall information and biomarker data

[1] Calculated from What We Eat in America 2003–4 (mean age 7.5 years). www.cdc.gov/nchs/NHANES

[2] NC: Not calculated

* ANOVA F test:, $p < 0.05$

Table 2

Consumption of major and minor food groups among 1101 BCERP girls (2004–2007).[1]

Food groups	Mean (Std) Servings per day
Total Grains	5.56 (1.69)
Grains, flour and dry mixes[2]	1.14 (1.01)
Rice	0.21 (0.30)
Flour in mixed foods	0.25 (0.33)
Bread	2.26 (1.13)
Pasta	0.58 (0.63)
Ready-made cereal	0.60 (0.54)
Cakes, pastries	0.47 (0.46)
Total Dairy	1.79 (0.83)
Whole fat dairy	0.56 (0.52)
Low fat dairy	1.22 (0.76)
Total Vegetables	1.48 (0.84)
Fresh vegetables	0.81 (0.65)
Non-fresh vegetables	
Processed	0.71 (0.51)
Canned	0.31 (0.35)
Total Fruit	2.09 (1.37)
Fresh fruit	0.83 (0.99)
Non-Fresh	
Processed	0.66 (0.58)
Canned	0.15 (0.25)
Total Meat	2.68 (1.38)
Non-poultry meat	1.50 (1.07)
Poultry	1.18 (0.99)
Poultry excluding fried chicken	0.79 (0.82)
Fried chicken: fast food, commercial entrees	0.39 (0.65)
Total fish and shellfish	0.28 (0.62)
Fresh	0.21 (0.51)
Non-Fresh	0.03 (0.12)
Canned	0.03 (0.12)
Fried, fast food	0.04 (0.18)
Eggs	0.23 (0.32)
Fats and oils	2.02 (1.29)
Total canned foods	0.51 (0.46)

[1] Includes NDSR food groupings and subgroups created to distinguish food packaging and processing that are used to examine associations with biomarkers.

[2] Average servings per day subgroups do not necessarily add up to the total group as only the main subgroups and foods are shown. Intended subgroups are all or some of the subgroups.

Table 3

Adjusted[1] geometric means (95% CI) of urinary phthalate and phenol biomarkers (μg/gC) in relation to intake of food groups and sub-groups[2] in 1101 girls (BCERP cohort 2004–7) for selected items showing associations with biomarkers (p<0.10).

FOOD GROUP		BPA (μg/gC)			
		Servings per day			
		0- < 0.5	0.5- <1.0	≥ 1.0	p- trend[3]
Grains, flour and dry mixes	GM (CI)	2.22 (1.98–2.50)	2.58 (2.28–2.93)	2.75 (2.51–3.03)	0.001
	N	312	261	501	
Flour in mixed foods[4,5]	GM (CI)	2.49 (2.29–2.71)	3.00 (2.57–3.51)	2.80 (2.17–3.62)	0.038
	N	890	138	46	
Total vegetables	GM (CI)	2.30 (1.93–2.74)	2.30 (2.03–2.61)	2.71 (2.48–2.96)	0.006
	N	82	220	772	
Non-fresh vegetables[5]	GM (CI)	2.36 (2.14–2.62)	2.68 (2.40–2.99)	2.81 (2.49–3.18)	0.010
	N	447	352	275	
Total poultry	GM (CI)	2.36 (2.10–2.65)	2.58 (2.27–2.93)	2.70 (2.45–2.97)	0.043
	N	292	253	529	
Poultry excluding fried/fast food[5,6]	GM (CI)	2.41 (2.18,2.67)	2.54 (2.25,2.87)	2.84 (2.54,3. 18)	0.011
	N	479	251	344	
Fats	GM (CI)	2.11 (1.69–2.64)	2.29 (1.99–2.63)	2.68 (2.46–2.92)	0.005
	N	64	181	829	
Total fish	GM (CI)	2.51 (2.30,2.73)	2.75 (2.21,3.43)	2.93 (2.48,3. 46)	0.050
	N	888	63	123	
Fried fish, fast food[5]	GM (CI)	2.54 (2.34–2.76)	3.28 (2.44–4.43)	3.83 (2.23–6.58)	0.028
	N	1030	34	10	

		ΣParabens (μg/gC)			
		Servings per day			
		0- < 0.5	0.5- <1.0	≥ 1.0	p- trend
Non-fresh vegetables	GM (CI)	99 (84–117)	103 (86–123)	78 (65–96)	0.046
	N	447	352	275	

ΣParabens (μg/gC)

		Servings per day			
		0- < 0.5	**0.5- <1.0**	**≥ 1.0**	**p- trend**
Poultry excluding fried/fast food[6]	GM (CI)	85 (72,100)	102 (83,125)	105 (87,127)	0.035
	N	479	251	344	

ΣDEHP (μg/gC)

		Servings per day			
		0- < 0.5	**0.5- <1.0**	**≥ 1.0**	**p- trend**
Grains, flour and dry mixes	GM (CI)	150 (133–169)	155 (137–176)	182 (166–200)	0.001
	N	312	261	501	
Rice[5]	GM (CI)	162 (148–177)	196 (167–230)	166 (116–237)	0.084
	N	924	127	23	
Whole Fat Dairy	GM (CI)	165 (151–181)	156 (135–173)	198 (172–228)	0.051
	N	611	277	186	
Total fish	GM (CI)	164 (150–178)	157 (126–196)	205 (173–242)	0.018
	N	888	63	123	
Fried fish, fast food[5]	GM (CI)	165 (152–179)	231 (171–312)	267 (154–460)	0.007
	N	1030	34	10	

mBzP(μg/gC)

		Servings per day			
		0- < 0.5	**0.5- <1.0**	**≥ 1.0**	**p- trend**
Grains, flour and dry mixes	GM (CI)	16.2 (11.5–23.0)	15.5 (12.6–18.8)	20.6 (18.8–22.7)	0.003
	N	312	261	501	
Flour from mixed foods[5]	GM (CI)	19.1(17.3–21)	23.0 (19.3–27.4)	22.5 (16.8–30.1)	0.040
	N	890	138	46	
Fresh Fruit[7]	GM (CI)	21.1 (18.8–23.6)	19.9 (17.4–22.8)	17.6 (15.4–20.1)	0.016
	N	467	295	312	

mCPP (μg/gC)					
		Servings per day			
		0- < 0.5	0.5- <1.0	≥ 1.0	p- trend
Fish: Fresh	GM (CI)	5.99 (5.56–6.45)	6.89 (5.64–8.41)	6.94 (5.82–8.26)	0.049
	N	930	61	83	

LowMWP (μg/gC)					
		Servings per day			
		0- < 0.5	0.5- <1.0	≥ 1.0	p- trend
Total poultry	GM (CI)	142(127,160)	144(127,163)	166(151,182)	0.008
	N	292	253	529	
Fried/processed chicken	GM (CI)	146(134,159)	175(154,200)	171(147,199)	0.005
	N	716	210	148	

[1] Adjusted for age at urine collection, race and caregiver education.

[2] Groups in Table 2.

[3] Trend test using median of each category as continuous variable

[4] Examples are flour in pizza, gravy.

[5] The number of girls by serving size in subgroups may be higher than the number in the total food group as the total food group included other foods, with more or less servings.

[6] Fried/processed chicken showed no association.

[7] The relationship is inverse.