Published in final edited form as:

Environ Res. 2016 October ; 150: 375–382. doi:10.1016/j.envres.2016.06.008.

The consumption of canned food and beverages and urinary Bisphenol A concentrations in NHANES 2003–2008

Jennifer C. Hartle^{a,*}, Navas-Acien^{b,c}, and Robert S. Lawrence^{b,d}

^aStanford Prevention Research Center in the Department of Medicine, Stanford University School of Medicine, 1070 Arastradero Road, Suite 100, Palo Alto, California, USA ^bDepartment of Environmental Health Sciences, Johns Hopkins University Bloomberg School of Public Health, Baltimore, 615 N. Wolfe St., Maryland, USA ^cDepartment of Environmental Health Sciences, Columbia University, Mailman School of Public Health, 722 West 168th St., New York, New York, USA ^dCenter for a Livable Future, Johns Hopkins University Bloomberg School of Public Health, 615 N. Wolfe St., Baltimore, Maryland, USA

Abstract

Background—Exposure to Bisphenol A (BPA) is ubiquitous and includes dietary and environmental pathways. BPA is rapidly glucuronidated in the body, and both BPA and its conjugates can be readily measured in urine.

Objectives—To investigate the contribution of canned food and beverages, known sources of BPA contamination, to BPA biomarkers of exposure using dietary and urinary BPA concentration information in a representative sample of the U.S. population.

Methods—We evaluated 7,669 NHANES 2003–2008 participants 6 years and older with 24-hour dietary recall information and urinary BPA concentrations available. Using linear regression models, we evaluated the associations between recent canned food and beverage consumption and urinary BPA concentrations, adjusting for potential confounders.

Results—We found 9% of our participants consumed one canned food in the past 24 hours and 2% consumed two or more canned foods. The consumption of one canned food vs. none was associated with 24% (95% CI 1.11, 1.38) higher urinary BPA concentrations. The consumption of two or more canned foods vs. none was associated with 54% (95% CI 1.27, 1.88) higher urinary BPA concentrations. The consumption of one or more of some specific types of canned foods vs. none were associated with higher urinary BPA concentrations: 41% (95% CI 1.23, 1.63) higher BPA for vegetable and fruit, 70% (95% CI 1.18, 2.44) higher for canned pasta, and 229% (95% CI

*Corresponding author.: 1070 Arastradero Road, Suite 100 Stanford Prevention Research Center. Palo Alto, CA 94304-1334. Tel 650-723-6254. Fax: 650-725-6906. jhartle@stanford.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Human Subjects

The study protocol was reviewed by the Johns Hopkins Bloomberg School of Public Health Institutional Review Board and deemed not to be human subjects research.

1.22, 4.30) higher for canned soup. Canned beverages were not associated with urinary BPA concentrations.

Conclusions—Canned food, including some specific types such as canned vegetable and fruit, canned pasta, and canned soup were associated with higher levels of urinary BPA concentrations.

Keywords

Bisphenol A; canned food; NHANES; diet; exposure

Background

Bisphenol A (BPA), a synthetic chemical with endocrine disrupting properties approved for use in food packaging in the 1960's (FDA 2015), is commonly utilized as a monomer base for polycarbonate plastic (PC) and as a linkage in epoxy resins. PC is a hard, clear plastic used in a wide range of consumer products including food storage containers (FDA 2015; Krishnan et al. 1993). Enamel coatings made of epoxy resins are used to line the inside of many food and beverage containers (Biles et al. 1997; FDA 2015). Additional BPA uses include as a color developer in thermal receipts (Biedermann et al. 2010; T. Geens et al. 2012; Liao and Kannan 2011), in medical devices (Tinne Geens et al. 2012), and as a component of some dental composites (Joskow et al. 2006; Olea et al. 1996).

The widespread use of polycarbonate plastics and epoxy resins in consumer products (Melzer et al. 2010; Tsai 2006; Willhite et al. 2008) has contributed to the ubiquitous BPA exposure in the human population, with BPA being detected in the urine of 92.6 % of the American population (Calafat et al. 2008). Although BPA exposure has been measured in household dust and air (Loganathan and Kannan 2011; Wilson et al. 2007) and in water contaminated by landfill leachate (Vandenberg et al. 2007; vom Saal et al. 2007), diet is considered the main contributor of BPA exposure (Christensen et al. 2015; Kang et al. 2006; von Goetz et al. 2010; Wilson et al. 2007).

BPA in food packaging is a concern due to its propensity to leach into the product. BPA molecules migrating into the food from its incomplete polymerization in the manufacturing of epoxy resins of cans was recognized in 1995 (Brotans et al. 1995). Subsequent studies have shown that BPA can leach into food from polycarbonate plastic bottles, plastic storage containers, and polyvinyl chloride (PVC) stretch film (Biles et al. 1997; Brede et al. 2003; Lopez-Cervantes and Paseiro-Losada 2003; McNeal et al. 2000; Noonan et al. 2011; Schecter et al. 2010; Yang et al. 2011). Intervention studies have shown an ability to decrease or increase urinary BPA concentration by controlling the consumption of known sources of BPA from the food system (Carwile et al. 2011; Rudel et al. 2011; Teeguarden et al. 2011). Studies closely following dietary intake and collecting multiple biomarker samples validated with pharmacokinetics data have shown that about two-thirds of BPA exposure can be attributed to diet, and one third is unaccounted for (Christensen et al. 2015). Analysis of fasting data have also shown that when dietary intake stops, BPA is still present in the body indicating non-food pathways or possible storage of BPA in fat (Stahlhut et al. 2009).

When BPA is ingested by humans, it is transformed by the liver into bisphenol A-glucuronide, a highly water soluble compound (Volkel et al. 2002). In pharmacokinetics studies, BPA's half-life in the body is less than six hours, and BPA is completely eliminated from the body in 24 hours (Volkel et al. 2002). Because of its rapid clearance from the body through urine, total urinary BPA compounds, comprised of free plus conjugated BPA, are the most appropriate BPA exposure assessment marker (Melzer et al. 2010). The best urinary collection method including single samples vs. 24-hour sampling, and the timing of sampling have been investigated (Arakawa et al. 2004; Christensen et al. 2012; Dekant and Volkel 2008; LaKind and Naiman 2015; Lassen et al. 2013; Mahalingaiah et al. 2008; Ye et al. 2011). The conclusions of those studies vary depending on a researcher's intent to determine individual or population level BPA exposures and short-term vs. long term exposure profiles. Spot urine samples collected by large cross-sectional population level surveys, such as the National Health and Nutrition Examination Survey (NHANES), can characterize average population exposures (Lakind and Naiman 2008; Ye et al. 2011).

The goal of this research was to evaluate the dietary contributions to urinary BPA in a representative sample of the US population. Using NHANES data, we focused on the dietary information collected in the 24-hour exposure window prior to collection of a urine sample for BPA testing. The 24-hour dietary recall food coding provided identification on canned food and canned beverages, dietary components known to be BPA exposure sources, allowing the inclusion of those dietary sources of BPA in the current analyses.

Methods

Study population

This study utilizes three cycles of publicly available NHANES data: 2003–2004, 2005–2006, and 2007–2008 (CDC 2003, 2005, 2007c). NHANES is an ongoing cross-sectional survey of the non-institutionalized US population that employs a complex, multi-stage survey design. Total urinary BPA concentration was first measured in NHANES 2003–2004 in a random one-third subsample of the NHANES population six years and older as part of the environmental phenols panel. Each subsequent survey cycle has followed the same BPA sampling plan. For this analysis, we used data from 7699 persons six years and older who completed their NHANES 2003–2008 interviews and examinations, environmental phenol panel, and for whom BPA data were present. We excluded 355 with missing diet data, and 942 participants missing other relevant covariates, leaving 6372 participants for the study's models. The study population is similar to the overall 7669 population in participant characteristics such as age, sex, education and race/ethnicity [data not shown].

Biomonitoring measurements

Urinary samples were collected when the participant visited the Mobile Examination Center (MEC). From the MEC, the urine samples were frozen and shipped for BPA analysis to the Division of Environmental Health Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention (CDC 2004, 2007a).

The processing laboratories used a lab method especially designed by NHANES to be sensitive to measuring BPA and other environmental phenols on the panel in urine. The method utilized solid phase extraction (SPE) coupled on-line to high-performance liquid chromatography (HPLC) and tandem mass spectrometry (MS/MS). They also used isotopically labeled internal standards to detect relatively low levels of phenols for 100 μ L of urine (CDC 2007b). The detection limits within an NHANES cycle were consistent. For the 2003–2004 cycle, the limit of detection (LOD) was 0.36 ng/ml. For 2005–2006 and 2007–2008, the LOD was 0.4 ng/ml. The percent of samples below the LOD was 6.5% for NHANES 2003–2004, 7.1% for NHANES 2005–2006 and 6.4% for NHANES 2007–2008 (CDC 2007b, 2009b, 2011). NHANES represented values below the limit of detection with the calculation $LOD/2$ (CDC 2009a).

Dietary Measures

On the same day as the urinary sample for BPA analysis was collected, the 24-hour dietary recall data were recorded by an interviewer during the MEC appointment (CDC 2002). These data were called “Day 1” to distinguish them from the second day of dietary recall information that was recorded from a phone interview 3 – 10 days after the MEC appointment. Since BPA is rapidly glucuronidated and cleared in the urine in approximately 24 hours (Nachman et al. 2014; Volkel et al. 2002), Day 1 24-hour dietary recall is an appropriate time to evaluate the associations between potential BPA food exposure and urinary BPA concentrations.

Following the 24-hour dietary recall, the dietary data are coded into food items using the USDA Food Codes (CDC 2006). USDA Food Codes are adjusted with every two-year NHANES cycle; removing codes that are underutilized and adding new codes for new commonly consumed food products. To analyze the association of consumption of food potentially high in BPA with urinary BPA concentrations, we re-coded the USDA Food Codes into categories based on the supporting information provided by the USDA Food Code fields entitled “main description” and “additional description” (USDA 2006, 2008, 2010). The food re-coding categories were: (1) foods that are definitely canned as worded in their description, (2) foods that are possibly canned, (3) canned beverages, and (4) foods that are not canned. Within the canned food codes, there was further categorization into five canned food groups based upon categories utilized in published studies (Bureau of Chemical Safety 2010; Cao et al. 2011; Holmes et al. 2005) of BPA and canned food. These canned food groups included: dairy, meat and fish, vegetable and fruit, soup, and pasta. For more details on which canned foods were placed in each food group, see Supplemental Material, Table 1. Canned beverages were not readily recognized with food code descriptions. In order to identify canned beverages from the dietary recall, USDA Food and Nutrient Database for Dietary Studies (FNDDS) food weights and food portion descriptions were utilized (USDA 2006, 2008, 2010). Portion descriptions identified the canned drinks and the associated portion code could link these descriptions to USDA Food Codes. (Moshfegh A, personal communication).

Re-coding of the USDA Food Codes reaped the definitely canned food numbers of 265, 257, and 258 for NHANES 2003–2004, 2005–2006, and 2007–2008, respectively. We reserved

the “definitely canned” grouping for foods definitively stated as being canned in the main and additional descriptions of the USDA Food Codes. In cases where a canned food was combined with another food packaging type into one food code, dietary components identified by these types of food codes had to be placed in the “possibly canned” food category for this research. For food codes that could possibly be canned, the corresponding numbers for each cycle were 137, 134, and 134, respectively.

Supplemental Table 2 shows the number of food codes that were identified by the re-coding process per canned food category. It also displays the number of times an item in a canned food category was reported to be eaten. The use of the term “item” in this table relates to the number of eating events a participant reported for a certain food and does not correspond to a specific portion size. Canned vegetable and fruit represent the majority of the canned food items that were identified, as well as the number of canned items that were consumed. The top three canned food groups eaten were canned vegetable and fruit, accounting for 50% of the canned food eaten, followed by canned meat and fish at 33%, and canned pasta at 8%.

Statistical analysis

Statistical analyses were performed with Stata (Stata 12.1 College Station, Texas). Statistical analysis accounted for the NHANES complex sampling design using strata, primary sampling units and specific sample weights for the BPA subsample following NHANES guidelines (CDC 2013). Standard errors were estimated using the Taylor series method of linearization to take into account stratification and clustering in “masked variance pseudo-psu” and “pseudo-stratum” variables in the NHANES dataset.

We used simple and multiple linear regressions on natural log-transformed urinary BPA concentrations to evaluate the association between canned foods and BPA exposure. In these models, the exponentiation of the beta coefficient corresponds to the geometric mean ratio of urinary BPA concentrations per unit increase in canned food consumption. The covariates selected for adjustment were informed by previous BPA exposure studies in NHANES that described relevant possible confounders based on sociodemographic and lifestyle characteristics (Calafat et al. 2008; Lakind and Naiman 2011; Nelson et al. 2012). They included gender, age, race, income, education, and smoking. We also included creatinine as a covariate to account for urine dilution (Barr et al. 2005) and included it in our models as a continuous variable. Before adding urine creatinine concentrations into the regression models, the creatinine concentration was normalized by taking the square root. This is one recommended method to allow for BPA concentrations to be adjusted for urinary creatinine while still allowing the other covariates in the model to remain independent of urinary creatinine concentration fluctuations (Barr et al. 2005).

The study population was grouped into five age categories: 6–11 years, 12–19 years, 20–39 years, 40–59 years, and 60 years and up. These age categories allowed comparability of this research to previous studies (Calafat et al. 2008; Lakind and Naiman 2008) and a roughly even distribution of the population among the age groups. Race and ethnicity were based on self-report and subsequently categorized by NHANES into five categories of Non-Hispanic White, Non-Hispanic Black, Mexican Americans, Other Hispanic, and Other ethnicity including Mixed Race in the analysis. We preserved as much information about the race/

ethnic categories as publicly available in NHANES to potentially inform future public health interventions.

For participants 20 years and older, we used self-reported education data and categorized it into four groups: less than high school, high school graduate or equivalent, some college, and college graduate and above. For participants 6–19 years of age, we used the education level of the head of household. For income, we used the poverty income ratio (PIR). Income category divisions were decided based upon the poverty income ratio cut points used for the public food assistance programs Supplemental Nutrition Assistance Program (SNAP) and the National School Lunch Program (NSLP) (USDA 2003, 2015). The income groupings were PIR 0.000–1.3000; 1.301 – 3.501, and 3.501 and above (CDC 1996).

To assess smoking exposure, including both active smoking and secondhand smoke exposure, we utilized the serum cotinine values, a specific tobacco biomarker, as follows: unexposed (serum cotinine < 0.011 ng/ml, the limit of detection [LOD]); exposure to secondhand smoke (LOD 0.011 – 10 ng/ml); and active smokers (≥ 10 ng/ml) (Avila-Tang et al. 2013). Adjusting for smoking is important as published research found associations between smoking and urinary BPA concentrations (Braun et al. 2011; He et al. 2009). BPA can be a substantial component of cigarette filters making BPA exposure an issue for those exposed from active smoking or secondhand smoke (Jackson and Darnell 1985).

Results

After recoding the 24-hour dietary recalls into canned food categories, 11% of participants reported consuming one or more canned food and 17% reported consuming one or more canned beverages. When combining definitely and possibly canned food consumption, 41% consumed at least one or more canned food (Table 2). The mean number of definitely canned foods and beverages consumed per day was 0.40 (Table 1). Canned food and beverage consumption was higher in men than women; by age was highest in young adults; by race/ethnicity was higher in non-Hispanic Whites, especially compared to other Hispanic and to other races; and was lower in participants with the lowest income group, in participants with lowest and highest education, and among participants not exposed to secondhand smoke.

After multivariable adjustment, increasing canned foods consumption was associated with higher urinary BPA concentration for the whole population, for the adult population, and for children (Table 2). Overall, compared to no definitely canned food consumed in the past 24 hours, the adjusted GM ratio (95% CI) for urinary BPA concentrations was 1.24 (1.11 – 1.38) for one canned food and 1.54 (1.27 – 1.88) for two canned foods or more (p-trend <0.001). The corresponding GM ratios for definitely and possibly canned foods were 1.12 (1.04 – 1.20) and 1.24 (1.12 – 1.37) (p-trend <0.001). For specific foods, the GM ratio (95% CI) of urinary BPA for consumption of one or more canned food versus none was 1.41 (CI 1.23, 1.63) for canned vegetable and fruit, 1.70 (1.18, 2.44) for canned pasta, and 2.29 (1.22, 4.30) for canned soup (Table 3). Canned beverages were not associated with urinary BPA concentrations.

The associations between canned food and urinary BPA concentrations were stronger among children compared to adults, with GM ratios (95% CI) comparing one and two or more definitely canned foods vs. no canned food equal to 1.33 (1.09 – 1.62) and 1.75 (1.14 – 2.70), respectively, for youth, and 1.21 (1.07, 1.37) and 1.49 (1.18, 1.89), respectively, for adults (Table 2). For specific canned foods, the youth and adult models showed a significant association between BPA concentration and consumption of canned vegetable and fruit with a GM ratio (95% CI) of 1.57 (1.21, 2.02) for youth and 1.37 (1.17, 1.60) for adults, and for canned pasta 1.62 (1.36, 1.93) and 1.68 (1.00, 2.81) for youth and adults, respectively (Table 3). The two age groups differ in their GM ratios for soup with the youth GM ratio not achieving statistical significance (1.41 (0.66, 3.03)).

Discussion

In this representative sample of the US population, canned food consumption in the last 24 hours was associated with higher urinary BPA concentrations in both children and adults. The association was stronger for children. Canned beverages were not associated with higher urinary BPA concentrations. Among specific canned foods, canned vegetable and fruit, canned pasta, and canned soup were associated with higher BPA concentrations while canned dairy and canned meat and fish were not. The association was strong for canned soup for the overall population, however, the strong association of urinary BPA concentration and soup consumption did not hold for the youth population (GM ratio (95% CI) of 1.41 (0.66, 3.03)). These findings need to be interpreted cautiously as the number of participants consuming canned soup was small (n=23 overall, n=8 for youth) with soup only representing 3% of the canned food eaten. This study is the first known analysis of NHANES data for dietary contributions of BPA exposure using USDA Food Codes. A major strength of the study includes the use of a nationally representative sample to evaluate associations between sources of exposure and biomarkers of internal dose.

One of the biggest challenges in this research was accurately identifying canned food from the dietary recall. The USDA Food Codes applied to the dietary recall are designed to capture the foods eaten for nutritional analysis. Their ability to be used for environmental health purposes is problematic because of the placement of several similar food items with different food packaging into the same food code. For example, one food code description of peaches is “peach, not specified as to raw, cooked, canned, frozen, or dried”. This means when dietary recalls are coded, the self-reporting of peaches eaten in any of these five forms will be placed into the same food code. As a result of this method, capturing the consumption of canned fruit, amongst other foods, was challenging. The conservative approach we applied of only using “definitely canned” for foods definitively stated as being canned in the main and additional descriptions of the USDA Food Codes with no overlap with other food packaging types might have missed a number of canned foods, but it is a more specific definition. The “definitely and possibly” food category which included canned food combined with another food packaging type into one food code is less specific and although the number of participants consuming canned foods is larger, and thus increasing power, the associations may have resulted in an underestimation of the associations. With both approaches we found statistically significant associations, although the strength of the association was stronger for the conservative definition, consistent with a more specific

definition. Overall, this study was successful in developing a method for identifying canned food and canned beverages from NHANES 24-hour dietary recall data using the USDA Food Codes and packaging information from the FNDDS files.

Twenty-four-hour dietary recall data have some limitations, most of which are accounted for with the NHANES sample design and our research purpose of investigating population level trends. This type of dietary recall data cannot be used to represent long-term intake as dietary intake is highly variable from day to day and by the day-of-the-week. Twenty-four-hour dietary recalls also rely on self-reported data which may not be accurate. The NHANES data collection works to account for these shortcomings by collecting dietary data on different days of the week and to collect data from a representative sample of the U.S. population, so that the resulting population level data can be used to assess group trends (Willett 1990).

Some food categories were more easily identified by the canned coding methodology. Canned vegetables were identified by over 140 food codes, leading to the reporting of 443 instances or 47% of canned food consumed by the sub-population analyzed (Supplemental Table 2). This is a more precise reflection of the potential for canned vegetable consumption in the United States as the USDA has calculated that 24% of the total vegetables available for consumption are canned (Buzby et al. 2010). For fruit, the USDA has estimated that 6% of all fruit available for consumption is canned (Buzby et al. 2010). We identified only 25 instances of canned fruit consumption in the overall population, and only 3 instances in the sub-population modeled, a probable underestimation of actual consumption.

We compared the trends in BPA exposure from canned food and beverage exposure to prior research. There is extensive literature investigating BPA contamination of food and beverages, developing the best analytical methods for detection and determining the factors influencing BPA migration, with some references listed here (Bureau of Chemical Safety 2010; Cao et al. 2009, 2010, 2011; Goodson et al. 2002; Imanaka et al. 2001; Lorber et al. 2015; Munguia-Lopez et al. 2005; Noonan et al. 2011; Sajiki et al. 2007; Schecter et al. 2010; Thomson and Grounds 2005; Yonekubo et al. 2008; Yoshida et al. 2001). For the following discussion, we compared our findings to research conducted in North America to provide a closer comparison to the foods purchased and consumed in the United States (Bureau of Chemical Safety 2010; Cao et al. 2009, 2010; Cao et al. 2011; Lorber et al. 2015; Noonan et al. 2011; Schecter et al. 2010). Although there is considerable variability in the BPA concentrations between foods and within food types (Noonan et al. 2011), we expected our population data would reflect the following trends previously identified in the literature: BPA concentration (ng/g) is lower in canned beverages than canned foods (Cao et al. 2009, 2010), canned vegetables, specifically green beans, peas, and creamed corn can have high BPA concentration values (Bureau of Chemical Safety 2010; Cao et al. 2010; Cao et al. 2011; Lorber et al. 2015; Noonan et al. 2011; Schecter et al. 2010), and canned tuna and canned soup, especially condensed soup, tend to have higher BPA concentration values (Bureau of Chemical Safety 2010; Cao et al. 2010; Cao et al. 2011). We found most of these trends to be present in our models. Canned beverages did not have an association with urinary BPA concentrations (p-trend = 0.44). The consumption of more canned vegetables with a GM ratio (95% CI) of 1.42 (1.23 – 1.63) and canned soup with 2.29 (1.22, 4.30) were

associated with higher urinary BPA concentrations. An unexpected trend found in our data was for canned fruit. Our initial analysis separated canned fruit into its own category due to the documented difference in can linings for fruit (Noonan et al. 2011; Oldring and Nehring 2007). The industry practice, confirmed by Noonan's laboratory, is for epoxy-phenolic coating to be absent on fruit can side walls, although there are sometimes coatings on the ends of the cans. Our model of canned fruit alone (not presented in Table 3), had a GM ratio (95% CI) of 1.23 (1.09, 1.39) for the consumption of one or more canned fruit versus none. This fruit model is based on a very small number of items (n=5), so this result may not be reflective of a real trend. For some analyses that stratified the data (e.g. the MEC appointment time sensitivity analysis) we could not determine the effect estimate for canned fruit only and we had to combine it with canned vegetables. For the same statistical reasoning, we combined tuna with meat in our final models. Our initial analysis separated canned tuna into its own category due to the reported high concentrations of BPA (Bureau of Chemical Safety 2010; Cao et al. 2010). Canned tuna alone (not presented in Table 3), had a GM ratio (95% CI) of 1.62 (1.13, 2.35), a positive association that was masked by our grouping with meat.

We considered the possible difference in urinary BPA concentration and canned food and beverage consumption by MEC appointment times. We conducted a sensitivity analysis comparing morning appointments (n=3605), afternoon appointments (n=2656), and evening appointments (n=1408). We found that the same trends existed for the morning and the afternoon appointments for the overall populations for all canned categories except for soup with a GM ratio (95% CI) of 1.77 (0.96, 3.25) for consumption of one or more servings of soup versus none for the morning appointments. The evening appointments exhibited the most deviation from the overall population trends, with canned food GM ratio (95% CI) of 1.19 (0.97, 1.47), canned food and beverages with 1.04 (0.91, 1.19), canned pasta with 0.81 (0.43, 1.53), and canned soup with 1.96 (0.77, 5.02) no longer being statistically significant. One explanation for this change in trend is that the evening appointment urinary BPA concentrations most likely reflected more recent consumption of BPA containing foods than food eaten on the previous day, as recorded in the 24-hour dietary recall.

To improve the dietary recall data's ability to be used for environmental health food packaging studies, utilization of the USDA Food Codes Standard Reference (SR) Links may be a good starting point (USDA 2010). SR links describe the components of a food that were used to calculate nutritional analysis. For example, the food code 28340660 for "chicken or turkey vegetable soup, home recipe" is linked to 12 different SR items. These SR links often include canned food that would have been useful for canned food consumption analysis. The SR codes are rich with canned food links to USDA Food Codes: for 2003–2004, 1162 canned food codes; for 2005–2006- 1130; and for 2007–2008- 1312 [data not shown]. In the case of chicken or turkey vegetable soup, this food code is linked to "tomato juice, canned, with salt," SR code number 11540. The recipes associated with a food code can only be used for nutritional analysis and do not necessarily reflect what people actually consumed. SR codes for canned foods cannot be used to determine packaging. A preliminary study of SR links tested regression models that include the SR codes for canned foods into the analysis. No statistically significant associations were discovered, affirming the inability to use the SR codes for food packaging studies. If dietary recall data collected more information about

food packaging similar to the details provided in SR codes, it might be possible to identify additional sources of canned food.

Our study using NHANES 2003–2008 data captures a crucial window of time in the use of BPA in the food system. BPA was authorized for use in all food applications at the time of the 2003–2008 NHANES utilized in this research. Starting in 2008, the BPA landscape started to shift more dramatically. In April 2008, Health Canada declared BPA to be a “dangerous substance” (Buka et al. 2009). Soon after, major manufacturers of polycarbonate plastic bottles abandoned the use of BPA and large retailers, such as Wal-Mart and Toys-R-Us, announced the phase-out of BPA containing baby bottles. May of this same year, the United States Congress requested that BPA be removed from infant formula packaging due to its health hazards. States started to introduce legislation to limit uses of BPA in food packaging in 2008 and, by 2009, over 20 states had introduced legislation to ban BPA in baby bottles and sippy cups (Houlihan et al. 2011; NCSL 2015). By January 2010, FDA had released a report expressing “some concern” for BPA in children’s products and cans. As BPA was being removed from packaging, there was an increase in the use of alternative bisphenols such as Bisphenol F (BPF) and Bisphenol S (BPS) that could be measured in humans (LaKind and Naiman 2015; Liao and Kannan 2013; Ye et al. 2015). Overall, the largest changes in BPA applications were in infant and toddler products, products not used by our sampled population of six years and older, with the official legislation to remove the use approval for BPA in PC baby bottles and sippy cups and in infant formula packaging occurring in 2012 and 2013 (FDA 2012, 2013). Our sampled time period and population could have been affected by the decision by some canned food manufacturers and reusable water bottle manufacturers to use BPA alternatives. NHANES environmental phenol testing does not measure bisphenol analogues, so overall bisphenol exposure analyses cannot be conducted.

A future research recommendation for NHANES would be to enhance the current dietary and urinary biological data collection system. The first morning void should be collected on the same day that the 24-hour dietary recall is performed. Research has shown that this urinary sample would likely be capturing the previous night’s dinner exposure to BPA (Teeguarden et al. 2011). The short time frame between consumption and dietary recall collection could also improve the accuracy of the food consumption reported and enhance researchers’ ability to characterize BPA dietary exposure. As analogues of BPA are currently not measured in NHANES, future efforts to sample for BPA and its alternatives in packaging including BPF and BPS are needed to understand the current state of overall bisphenol exposure in the U.S. population. These suggested expansions in the NHANES data collection could be an important step in strengthening epidemiologic data about BPA exposures.

Conclusions

This analysis of NHANES dietary recall data showed that canned foods contribute to BPA exposure in the population of the United States, including both children and adults. Urinary BPA concentrations increased with the number of canned foods, and specifically with consumption of canned vegetable and fruit, pasta, and soup. Most of these trends mirror

findings of laboratory studies that directly measured BPA concentration of canned food and beverages and confirm the importance of banning the use of BPA from those products to prevent dietary exposure to BPA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

JH wished to acknowledge the ongoing support of her co-authors and the Center for a Livable Future at Johns Hopkins University Bloomberg School of Public Health.

Grant Support

This investigation was supported by funds from the Center for a Livable Future-Lerner Doctoral Fellowship, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD and a National Institutes of Health Grant T32 HL007034.

Abbreviations

NHANES	National Health and Nutrition Examination Survey
BPA	Bisphenol A
GM	Geometric Mean
CI	Confidence Interval

References

- Arakawa C, Fujimaki K, Yoshinaga J, Imai H, Serizawa S, Shiraishi H. Daily urinary excretion of bisphenol A. *Environ Health Prev Med*. 2004; 9:22. [PubMed: 21432334]
- Avila-Tang E, Al-Delaimy WK, Ashley DL, Benowitz N, Bernert JT, Kim S, et al. Assessing secondhand smoke using biological markers. *Tobacco Control*. 2013; 22:164–171. [PubMed: 22940677]
- Barr DB, Wilder LC, Caudill SP, Gonzalez AJ, Needham LL, Pirkle JL. Urinary creatinine concentrations in the U.S. Population: Implications for urinary biologic monitoring measurements. *Environ Health Perspect*. 2005; 113:192. [PubMed: 15687057]
- Biedermann S, Tschudin P, Grob K. Transfer of bisphenol A from thermal printer paper to the skin. *Analytical and Bioanalytical Chemistry*. 2010; 398:571–576. [PubMed: 20623271]
- Biles JE, McNeal TP, Begley TH, Hollifield HC. Determination of bisphenol-A in reusable polycarbonate food-contact plastics and migration to food-simulating liquids. *Journal of Agricultural and Food Chemistry*. 1997; 45:3541.
- Braun JM, Kalkbrenner AE, Calafat AM, Bernert JT, Ye X, Silva MJ, et al. Variability and predictors of urinary bisphenol A concentrations during pregnancy. *Environ Health Perspect*. 2011; 119:131. [PubMed: 21205581]
- Brede C, Fjeldal P, Skjevrak I, Herikstad H. Increased migration levels of bisphenol A from polycarbonate baby bottles after dishwashing, boiling and brushing. *Food Addit Contam*. 2003; 20:684. [PubMed: 12888395]
- Brotons JA, Olea-Serrano MF, Villalobos M, Pedraza V, Olea N. Xenoestrogens released from lacquer coatings in food cans. *Environ Health Perspect*. 1995; 103:608. [PubMed: 7556016]

- Buka I, Osornio-Vargas A, Walker R. Canada declares bisphenol A a 'dangerous substance': Questioning the safety of plastics. *Paediatrics & Child Health*. 2009; 14:11–13. [PubMed: 19436577]
- Bureau of Chemical Safety. Survey of bisphenol A in canned food products from Canadian markets. Ottawa, ON: Health Canada; 2010. 978-1-100-16029-0
- Buzby, JC.; Wells, HF.; Kumcu, A.; Lin, BH.; Lucier, G.; Perez, A. Canned fruit and vegetable consumption in the United States. Beltsville, Maryland: USDA Economic Research Service; 2010.
- Calafat AM, Ye X, Wong LY, Reidy JA, Needham LL. Exposure of the U.S. Population to bisphenol A and 4-tertiary-octylphenol: 2003–2004. *Environ Health Perspect*. 2008; 116:39. [PubMed: 18197297]
- Cao XL, Corriveau J, Popovic S. Levels of bisphenol A in canned soft drink products in Canadian markets. *Journal of Agricultural and Food Chemistry*. 2009; 57:1307. [PubMed: 19170636]
- Cao XL, Corriveau J, Popovic S. Bisphenol A in canned food products from Canadian markets. *Journal of Food Protection*. 2010; 73:1085. [PubMed: 20537264]
- Cao XL, Perez-Locas C, Dufresne G, Clement G, Popovic S, Beraldin F, et al. Concentrations of bisphenol A in the composite food samples from the 2008 Canadian total diet study in Quebec city and dietary intake estimates. *Food Addit Contam Part A Chem Anal Control Expo Risk Assess*. 2011; 28:791.
- Carwile JL, Ye X, Zhou X, Calafat AM, Michels KB. Canned soup consumption and urinary bisphenol A: A randomized crossover trial. *JAMA*. 2011; 306:2218. [PubMed: 22110104]
- CDC (Centers for Disease Control). Analytic and reporting guidelines: The Third National Health and Nutrition Examination Survey, NHANES III 1988–94. 1996. Available: <http://www.cdc.gov/nchs/data/nhanes/nhanes3/nh3gui.pdf> [accessed February 3, 2015]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey: MEC in-person dietary interviewers procedures manual. 2002. Available: http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/DIETARY_MEC.pdf [accessed February 3, 2015]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey: NHANES 2003–2004. 2003. Available: http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/nhanes03_04.htm [accessed February 3, 2015]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey: Laboratory Procedures Manual. 2004. Available: http://www.cdc.gov/nchs/data/nhanes/nhanes_03_04/lab.pdf [accessed April 28, 2016]
- CDC (Centers for Disease Control). National Health Nutrition Examination Survey NHANES 2005–2006. 2005. Available: http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/nhanes05_06.htm [accessed February 3, 2015]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey dietary interview - individual foods, first day (dr1iff_c) data documentation, codebook, and frequencies. 2006. Available: http://wwwn.cdc.gov/nchs/nhanes/2003-2004/DR1IFF_C.htm#Component_Description [accessed April 25, 2016]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey: 2002–2004 laboratory quality assurance and monitoring. 2007a. Available: https://wwwn.cdc.gov/Nchs/Nhanes/2003-2004/L24EPH_C.htm#Laboratory_Quality_Assurance_and_Monitoring [accessed April 25, 2016]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey 2003 – 2004: Environmental phenols analytic notes. 2007b. Available: http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/L24EPH_C.htm#Analytic_Notes [accessed February 3, 2015]
- CDC (Centers for Disease Control). National Health and Nutrition Examination Survey-NHANES 2007–2008. 2007c. Available: http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/nhanes07_08.htm [accessed February 3, 2015]
- CDC (Centers for Disease Control). Fourth National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: U.S. Department of Health and Human Services Centers for Disease Control and Prevention; 2009a.

- CDC (Centers for Disease Control). NHANES 2005 – 2006: Environmental phenols and parabens analytic notes. 2009b. Available: http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/EPH_D.htm#Analytic_Notes [accessed February 3, 2015]
- CDC (Centers for Disease Control). NHANES 2007 – 2008: Environmental phenols analytic notes. 2011. Available: http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/EPH_E.htm#Analytic_Notes [accessed February 3, 2015]
- Christensen KL, Lorber M, Koch HM, Kolossa-Gehring M, Morgan MK. Population variability of phthalate metabolites and bisphenol A concentrations in spot urine samples versus 24- or 48-h collections. *J Expo Sci Environ Epidemiol*. 2012; 22:632–640. [PubMed: 22669498]
- Christensen KL, Lorber M, Ye X, Calafat AM. Reconstruction of bisphenol A intake using a simple pharmacokinetic model. *J Expo Sci Environ Epidemiol*. 2015; 25:240–248. [PubMed: 24252884]
- Dekant W, Volkel W. Human exposure to bisphenol A by biomonitoring: Methods, results and assessment of environmental exposures. *Toxicol Appl Pharmacol*. 2008; 228:114. [PubMed: 18207480]
- FDA (Food and Drug Administration). Federal Register, volume 77 issue 137 (July 17, 2012). Final Rule. Fed Reg 77 FR 41899. 2012
- FDA (Food and Drug Administration). Federal Register, volume 78 issue 134 (July 12, 2013). Final Rule. Fed Reg 78 FR 1840. 2013
- FDA (Food and Drug Administration). Bisphenol A (BPA): Use in food application. 2015. Available: <http://www.fda.gov/NewsEvents/PublicHealthFocus/ucm064437.htm#background> [accessed April 24, 2015]
- Geens T, Aerts D, Berthot C, Bourguignon J-P, Goeyens L, Lecomte P, et al. A review of dietary and non-dietary exposure to bisphenol-A. *Food and Chemical Toxicology*. 2012; 50:3725–3740. [PubMed: 22889897]
- Geens T, Goeyens L, Kannan K, Neels H, Covaci A. Levels of bisphenol-A in thermal paper receipts from Belgium and estimation of human exposure. *Sci Total Environ*. 2012;435–436. 30. [PubMed: 23026150]
- Goodson A, Summerfield W, Cooper I. Survey of bisphenol A and bisphenol F in canned foods. *Food Addit Contam*. 2002; 19:796. [PubMed: 12227943]
- He Y, Miao M, Herrinton LJ, Wu C, Yuan W, Zhou Z, et al. Bisphenol A levels in blood and urine in a Chinese population and the personal factors affecting the levels. *Environ Res*. 2009; 109:629–633. [PubMed: 19426969]
- Holmes MJ, Hart A, Northing P, Oldring PK, Castle L, Stott D, et al. Dietary exposure to chemical migrants from food contact materials: A probabilistic approach. *Food Addit Contam*. 2005; 22:907–919. [PubMed: 16236636]
- Houlihan, J.; Lunder, S.; Jacob, A. Timeline: BPA from invention to phase-out. 2011. Available: <http://www.ewg.org/research/timeline-bpa-invention-phase-out> [accessed April 25, 2016]
- Imanaka M, Sasaki K, Nemoto S, Ueda E, Murakami E, Miyata D, et al. Determination of bisphenol A in foods using GC/MS. *Shokuhin Eiseigaku Zasshi*. 2001; 42:71. [PubMed: 11486386]
- Jackson, W.; Darnell, W. Process for foaming cellulose acetate rod. US Patent. 4,507,256. 1985. filed 26, May 1983, and issued 26 March 1985
- Joskow R, Barr DB, Barr JR, Calafat AM, Needham LL, Rubin C. Exposure to bisphenol A from bis-glycidyl dimethacrylate-based dental sealants. *J Am Dent Assoc*. 2006; 137:353. [PubMed: 16570469]
- Kang JH, Kondo F, Katayama Y. Human exposure to bisphenol A. *Toxicology*. 2006; 226:79. [PubMed: 16860916]
- Krishnan AV, Stathis P, Permuth SF, Tokes L, Feldman D. Bisphenol-A: An estrogenic substance is released from polycarbonate flasks during autoclaving. *Endocrinology*. 1993; 132:2279. [PubMed: 8504731]
- Lakind JS, Naiman DQ. Bisphenol A (BPA) daily intakes in the United States: Estimates from the 2003–2004 NHANES urinary BPA data. *J Expo Sci Environ Epidemiol*. 2008; 18:608. [PubMed: 18414515]

- Lakind JS, Naiman DQ. Daily intake of bisphenol A and potential sources of exposure: 2005–2006 National Health and Nutrition Examination Survey. *J Expo Sci Environ Epidemiol*. 2011; 21:272. [PubMed: 20237498]
- LaKind JS, Naiman DQ. Temporal trends in bisphenol A exposure in the United States from 2003–2012 and factors associated with bpa exposure: Spot samples and urine dilution complicate data interpretation. *Environ Res*. 2015; 142:84–95. [PubMed: 26121292]
- Lassen TH, Frederiksen H, Jensen TK, Petersen JH, Main KM, Skakkebaek NE, et al. Temporal variability in urinary excretion of bisphenol A and seven other phenols in spot, morning, and 24-h urine samples. *Environ Res*. 2013; 126:164–170. [PubMed: 23932849]
- Liao C, Kannan K. Widespread occurrence of bisphenol A in paper and paper products: Implications for human exposure. *Environ Sci Technol*. 2011; 45:9372. [PubMed: 21939283]
- Liao C, Kannan K. Concentrations and profiles of bisphenol A and other bisphenol analogues in foodstuffs from the United States and their implications for human exposure. *J Agric Food Chem*. 2013; 61:4655–4662. [PubMed: 23614805]
- Loganathan SN, Kannan K. Occurrence of bisphenol A in indoor dust from two locations in the Eastern United States and implications for human exposures. *Arch Environ Contam Toxicol*. 2011; 61:68–73. [PubMed: 21221962]
- Lopez-Cervantes J, Paseiro-Losada P. Determination of bisphenol A in, and its migration from, PVC stretch film used for food packaging. *Food Addit Contam*. 2003; 20:596. [PubMed: 12881134]
- Lorber M, Schecter A, Paepke O, Shropshire W, Christensen K, Birnbaum L. Exposure assessment of adult intake of bisphenol A (BPA) with emphasis on canned food dietary exposures. *Environ Int*. 2015; 77:55–62. [PubMed: 25645382]
- Mahalingaiah S, Meeker JD, Pearson KR, Calafat AM, Ye X, Petrozza J, et al. Temporal variability and predictors of urinary bisphenol A concentrations in men and women. *Environ Health Perspect*. 2008; 116:173. [PubMed: 18288314]
- McNeal, TP.; Biles, JE.; Begley, TH.; Craun, JC.; Hopper, ML.; Sack, CA. Determination of suspected endocrine disruptors in foods and food packaging. In: Keith, L.; Jones-Lepp, TL.; Needham, LL., editors. *Analysis of environmental endocrine disruptors*. Washington, D.C.: American Chemical Society; 2000. p. 33
- Melzer D, Rice NE, Lewis C, Henley WE, Galloway TS. Association of urinary bisphenol A concentration with heart disease: Evidence from NHANES 2003/06. *PLoS One*. 2010; 5:e8673. [PubMed: 20084273]
- Munguia-Lopez EM, Gerardo-Lugo S, Peralta E, Bolumen S, Soto-Valdez H. Migration of bisphenol A (BPA) from can coatings into a fatty-food simulant and tuna fish. *Food Addit Contam*. 2005; 22:892. [PubMed: 16192075]
- Nachman RM, Hartle JC, Lees PS, Groopman JD. Early life metabolism of bisphenol A: A systematic review of the literature. *Current Environmental Health Reports*. 2014; 1:90–100. [PubMed: 25838989]
- NCSL (National Conference of State Legislatures). NCSL policy update: State restriction on bisphenol A (BPA) in consumer products. 2015. Available: <http://www.ncsl.org/research/environment-and-natural-resources/policy-update-on-state-restrictions-on-bisphenol-a.aspx> [accessed April 25, 2016]
- Nelson JW, Scammell MK, Hatch EE, Webster TF. Social disparities in exposures to bisphenol A and polyfluoroalkyl chemicals: A cross-sectional study within NHANES 2003–2006. *Environ Health*. 2012; 11:10. [PubMed: 22394520]
- Noonan GO, Ackerman LK, Begley TH. Concentration of bisphenol A in highly consumed canned foods on the U.S. Market. *Journal of Agricultural and Food Chemistry*. 2011; 59:7178. [PubMed: 21598963]
- Oldring, PKT.; Nehring, U. Metal packaging for foodstuffs. In: ILSI. , editor. *Packaging materials*. Belgium: 2007.
- Olea N, Pulgar R, Perez P, Olea-Serrano F, Rivas A, Novillo-Fertrell A, et al. Estrogenicity of resin-based composites and sealants used in dentistry. *Environ Health Perspect*. 1996; 104:298. [PubMed: 8919768]

- Rudel RA, Gray JM, Engel CL, Rawsthorne TW, Dodson RE, Ackerman JM, et al. Food packaging and bisphenol A and bis(2-ethylhexyl) phthalate exposure: Findings from a dietary intervention. *Environ Health Perspect.* 2011; 119:914. [PubMed: 21450549]
- Sajiki J, Miyamoto F, Fukata H, Mori C, Yonekubo J, Hayakawa K. Bisphenol A (BPA) and its source in foods in Japanese markets. *Food Addit Contam.* 2007; 24:103. [PubMed: 17164221]
- Schecter A, Malik N, Haffner D, Smith S, Harris TR, Paepke O, et al. Bisphenol A (BPA) in U.S. Food. *Environ Sci Technol.* 2010; 44:9425. [PubMed: 21038926]
- Stahlhut RW, Welshons WV, Swan SH. Bisphenol A data in NHANES suggest longer than expected half-life, substantial nonfood exposure, or both. *Environ Health Perspect.* 2009; 117:784. [PubMed: 19479022]
- Teeguarden JG, Calafat AM, Ye X, Doerge DR, Churchwell MI, Gunawan R, et al. Twenty-four hour human urine and serum profiles of bisphenol A during high-dietary exposure. *Toxicol Sci.* 2011; 123:48. [PubMed: 21705716]
- Thomson BM, Grounds PR. Bisphenol A in canned foods in New Zealand: An exposure assessment. *Food Addit Contam.* 2005; 22:65. [PubMed: 15895613]
- Tsai WT. Human health risk on environmental exposure to bisphenol-A: A review. *J Environ Sci Health C Environ Carcinog Ecotoxicol Rev.* 2006; 24:225. [PubMed: 17114111]
- USDA (United States Department of Agriculture). Child nutrition programs—income eligibility guidelines. 2003. Available: <http://www.fns.usda.gov/sites/default/files/IEGs03-04.pdf> [accessed April 27, 2016]
- USDA (United States Department of Agriculture). USDA Food and Nutrient Database for Dietary Studies, 2.0. Beltsville, MD: 2006.
- USDA (United States Department of Agriculture). USDA Food and Nutrient Database for Dietary Studies, 3.0. Beltsville, MD: 2008.
- USDA (United States Department of Agriculture). USDA Food and Nutrient Database for Dietary Studies, 4.1. Beltsville, MD: 2010.
- USDA (United States Department of Agriculture). Supplemental Nutrition Program (SNAP): Income (rules on income limits). 2015. Available: <http://www.fns.usda.gov/snap/income-rules-income-limits> [accessed April 26, 2016]
- Vandenberg LN, Hauser R, Marcus M, Olea N, Welshons WV. Human exposure to bisphenol A (BPA). *Reprod Toxicol.* 2007; 24:139. [PubMed: 17825522]
- Volkel W, Colnot T, Csanady GA, Filser JG, Dekant W. Metabolism and kinetics of bisphenol A in humans at low doses following oral administration. *Chem Res Toxicol.* 2002; 15:1281. [PubMed: 12387626]
- vom Saal FS, Akingbemi BT, Belcher SM, Birnbaum LS, Crain DA, Eriksen M, et al. Chapel Hill bisphenol A expert panel consensus statement: Integration of mechanisms, effects in animals and potential to impact human health at current levels of exposure. *Reprod Toxicol.* 2007; 24:131. [PubMed: 17768031]
- von Goetz N, Wormuth M, Scheringer M, Hungerbühler K. Bisphenol A: How the most relevant exposure sources contribute to total consumer exposure. *Risk Anal.* 2010; 30:473. [PubMed: 20136739]
- Willett, W. *Nutritional epidemiology*. New York: Oxford University Press; 1990.
- Willhite CC, Ball GL, McLellan CJ. Derivation of a bisphenol A oral reference dose (RfD) and drinking-water equivalent concentration. *J Toxicol Environ Health B Crit Rev.* 2008; 11:69. [PubMed: 18188738]
- Wilson NK, Chuang JC, Morgan MK, Lordo RA, Sheldon LS. An observational study of the potential exposures of preschool children to pentachlorophenol, bisphenol-A, and nonylphenol at home and daycare. *Environ Res.* 2007; 103:9. [PubMed: 16750524]
- Yang CZ, Yaniger SI, Jordan VC, Klein DJ, Bittner GD. Most plastic products release estrogenic chemicals: A potential health problem that can be solved. *Environ Health Perspect.* 2011; 119:989. [PubMed: 21367689]
- Ye X, Wong LY, Bishop AM, Calafat AM. Variability of urinary concentrations of bisphenol A in spot samples, first morning voids, and 24-hour collections. *Environ Health Perspect.* 2011; 119:983–988. [PubMed: 21406337]

- Ye X, Wong LY, Kramer J, Zhou X, Jia T, Calafat AM. Urinary concentrations of bisphenol A and three other bisphenols in convenience samples of U.S. Adults during 2000–2014. *Environ Sci Technol*. 2015; 49:11834–11839. [PubMed: 26360019]
- Yonekubo J, Hayakawa K, Sajiki J. Concentrations of bisphenol A, bisphenol A diglycidyl ether, and their derivatives in canned foods in Japanese markets. *Journal of Agricultural and Food Chemistry*. 2008; 56:2041. [PubMed: 18284199]
- Yoshida T, Horie M, Hoshino Y, Nakazawa H. Determination of bisphenol A in canned vegetables and fruit by high performance liquid chromatography. *Food AdditContam*. 2001; 18:69.

Highlights

- 9% participants consumed one canned food and 2% two or more on the previous day.
- One canned food vs. none was associated with 24% higher urinary BPA concentrations.
- 2 canned foods vs. none was associated with 54% higher urinary BPA concentrations.
- Some canned foods (vegetables, fruit, pasta, soup) were associated with higher BPA.
- Canned beverages were not associated with urinary BPA concentrations.

Urinary BPA Concentrations and Daily Canned Food and Beverage Consumption by Participant Characteristics in NHANES 2003–2008

Table 1

Characteristic	N (%)	GM (95% CI) of BPA (ng/ml)	p-value	Mean (95% CI) Daily Canned Food and Beverages	p-value
Sample Population	6372 (100)	2.17 (2.06, 2.28)	–	0.40 (0.37, 0.43)	–
GENDER					
Male	3167 (49.7)	2.36 (2.23, 2.50)	<0.001	0.41 (0.37, 0.46)	0.37
Female	3205 (50.3)	2.00 (1.86, 2.14)		0.39 (0.34, 0.43)	
AGE					
6 to 11	789 (12.4)	2.89 (2.61, 3.21)	<0.001	0.34 (0.27, 0.42)	0.66
12 to 19	1490 (23.4)	2.83 (2.65, 3.04)		0.35 (0.29, 0.40)	
20 to 39	1450 (22.8)	2.47 (2.28, 2.67)		0.45 (0.40, 0.50)	
40 to 59	1286 (20.1)	1.88 (1.73, 2.04)		0.40 (0.36, 0.45)	
60 +	1357 (21.3)	1.65 (1.53, 1.79)		0.37 (0.32, 0.42)	
RACE			0.90		<0.001
Non-Hispanic White	2759 (43.3)	2.08 (1.95, 2.21)		0.43 (0.39, 0.47)	
Non-Hispanic Black	1530 (24.0)	3.02 (2.78, 3.28)		0.37 (0.33, 0.41)	
Mexican-American	1462 (23.0)	2.18 (2.01, 2.36)		0.37 (0.31, 0.42)	
Other Hispanic	351 (5.5)	2.28 (1.91, 2.73)		0.22 (0.15, 0.29)	
Other Race/Multiracial	270 (4.2)	1.79 (1.45, 2.20)		0.27 (0.17, 0.38)	
INCOME			<0.001		0.67
PIR 0 – 1.300	2103 (33.0)	2.69 (2.48, 2.92)		0.39 (0.35, 0.43)	
PIR 1.301 – 3.500	2433 (38.2)	2.31 (2.15, 2.47)		0.40 (0.35, 0.45)	
PIR 3.501 – 5	1836 (28.8)	1.85 (1.73, 1.99)		0.40 (0.36, 0.45)	
EDUCATION			<0.001		0.41
Less than high school	1843 (28.9)	2.32 (2.14, 2.52)		0.38 (0.32, 0.43)	
High school grad	1548 (24.3)	2.25 (2.07, 2.44)		0.42 (0.37, 0.47)	
Some college	1796 (28.2)	2.37 (2.18, 2.58)		0.44 (0.40, 0.49)	
College grad +	1185 (18.6)	1.79 (1.62, 1.97)		0.34 (0.28, 0.40)	
SMOKING			<0.001		0.004
Not exposed	1165 (18.3)	1.62 (1.47, 1.80)		0.31 (0.26, 0.37)	
Env. Exposure	3933 (61.7)	2.33 (2.22, 2.45)		0.41 (0.37, 0.46)	

Characteristic	N (%)	GM (95% CI) of BPA (ng/ml)	p-value	Mean (95% CI) Daily Canned Food and Beverages	p-value
Active Smoker	1274 (20.0)	2.28 (2.10, 2.47)		0.43 (0.38, 0.49)	
BPA Quintiles			<0.001		<0.001
Q1	1284 (20.2)	0.47 (0.46, 0.49)		0.35 (0.29, 0.40)	
Q2	1334 (20.9)	1.36 (1.34, 1.38)		0.34 (0.29, 0.39)	
Q3	1248 (19.6)	2.38 (2.35, 2.41)		0.43 (0.36, 0.50)	
Q4	1270 (19.9)	4.10 (4.05, 4.16)		0.47 (0.39, 0.55)	
Q5	1236 (19.4)	10.7 (10.2, 11.2)		0.43 (0.37, 0.49)	

GM= geometric mean, CI = confidence interval, PIR = Poverty Income Ratio

Ratios of Urinary Bisphenol A Concentrations and Self-Reported Canned Food Type Intake in the past 24-hours-Main Canned Food Categories

Table 2

		Whole Population (n=6372)		Youth Pop. (6–19 yrs) (n=2279)		Adult Pop. (20–83 yrs) (n=4093)	
Overall Food Categories:		n	Ratio (95% CI)	n	Ratio (95% CI)	n	Ratio (95% CI)
Canned Food (Definitely)							
0		5696	1.00 (reference)	2073	1.00 (reference)	3623	1.00 (reference)
1		558	1.24 (1.11, 1.38)	173	1.33 (1.09, 1.62)	385	1.21 (1.07, 1.37)
2 or more		118	1.54 (1.27, 1.88)	33	1.75 (1.14, 2.70)	85	1.49 (1.18, 1.89)
p-value for trend			<0.001		<0.001		<0.001
Canned Beverages							
0		5309	1.00 (reference)	1963	1.00 (reference)	3346	1.00 (reference)
1		784	1.02 (0.95, 1.11)	233	1.17 (1.00, 1.36)	551	1.00 (0.91, 1.09)
2 or more		279	1.03 (0.91, 1.18)	83	1.06 (0.82, 1.38)	196	1.02 (0.88, 1.19)
p-value for trend			0.44		0.23		0.81
Canned Food (Definitely and Possibly)							
0		4030	1.00 (reference)	1474	1.00 (reference)	2556	1.00 (reference)
1		1776	1.12 (1.04, 1.20)	623	1.16 (1.04, 1.29)	1153	1.10 (1.02, 1.19)
2 or more		823	1.24 (1.12, 1.37)	273	1.43 (1.14, 1.79)	550	1.20 (1.07, 1.33)
p-value for trend			<0.001		0.001		<0.001
Canned Food and Beverages							
0		4746	1.00 (reference)	1782	1.00 (reference)	2964	1.00 (reference)
1		1167	1.11 (1.02, 1.21)	366	1.25 (1.06, 1.47)	801	1.08 (0.98, 1.19)
2 or more		459	1.20 (1.07, 1.33)	131	1.34 (1.07, 1.68)	328	1.16 (1.02, 1.32)
p-value for trend			<0.001		0.004		0.003

GM= geometric mean, CI = confidence interval

Whole, Youth, and Adult Population models shown as adjusted ratio of geometric means (95% CI), adjusted for sex, age, race, income, education, smoking, and creatinine.

Table 3

Ratios of Urinary Bisphenol A Concentrations and Self-Reported Canned Food Type Intake in the past 24-hours- Canned Food Sub-categories

Canned Food Sub-Categories.:	Whole Population (n=6372)				Youth Pop. (6–19 yrs) (n=2279)				Adult Pop. (20–83 yrs) (n=4093)			
	n	Ratio (95% CI)	p-value	n	Ratio (95% CI)	p-value	n	Ratio (95% CI)	p-value	n	Ratio (95% CI)	p-value
Veg and Fruit – 0	6021	1.00 (ref.)		2176	1.00 (ref.)		3845	1.00 (ref.)		3845	1.00 (ref.)	
Veg and Fruit - 1 or more	351	1.41 (1.23, 1.63)	<0.001	103	1.57 (1.21, 2.02)	0.001	248	1.37 (1.17, 1.60)	<0.001	248	1.37 (1.17, 1.60)	<0.001
Pasta- 0	6306	1.0 (ref.)		2246	1.00 (ref.)		4060	1.00 (ref.)		4060	1.00 (ref.)	
Pasta- 1 or more	66	1.70 (1.18, 2.44)	0.01	33	1.62 (1.36, 1.93)	<0.001	33	1.68 (1.00, 2.81)	0.05	33	1.68 (1.00, 2.81)	0.05
Soup- 0	6349	1.00 (ref.)		2271	1.00 (ref.)		4078	1.00 (ref.)		4078	1.00 (ref.)	
Soup- 1 or more	23	2.29 (1.22, 4.30)	0.01	8	1.41 (0.66, 3.03)	0.37	15	2.65 (1.23, 5.70)	0.01	15	2.65 (1.23, 5.70)	0.01
Dairy- 0	6327	1.00 (ref.)		2259	1.00 (ref.)		4068	1.00 (ref.)		4068	1.00 (ref.)	
Dairy- 1 or more	45	1.14 (0.72, 1.80)	0.57	20	1.00 (0.64, 1.55)	1.00	25	1.19 (0.64, 2.22)	0.57	25	1.19 (0.64, 2.22)	0.57
Meat and Fish- 0	6125	1.00 (ref.)		2217	1.00 (ref.)		3908	1.00 (ref.)		3908	1.00 (ref.)	
Meat and Fish- 1 or more	247	1.02 (0.86, 1.20)	0.84	62	1.02 (0.71, 1.46)	0.90	185	1.02 (0.87, 1.19)	0.85	185	1.02 (0.87, 1.19)	0.85

GM= geometric mean, CI = confidence interval, Veg = Vegetable, ref.= reference

Whole, Youth, and Adult Population models shown as adjusted ratio of geometric means (95% CI), adjusted for sex, age, race, income, education, smoking, and creatinine.