ENVIRONMENTAL RESEARCH
COMMUNICATIONS

LETTER • OPEN ACCESS

Prenatal bisphenol A (BPA) exposure in a Brooklyn study of Afro-Caribbean women

Environmental Research Communications

LETTER

Prenatal bisphenol A (BPA) exposure in a Brooklyn study of Afro-Caribbean women

Lori A Hoepner[1,6], **Rolf U Halden**[2,3], **Benny F G Pycke**[4], **Ovadia Abulafia**[5], **David M Sherer**[5] and **Laura A Geer**[1]

[1] Department of Environmental and Occupational Health Sciences, SUNY Downstate Health Sciences University, School of Public Health, Brooklyn, NY 11203, United States of America
[2] School of Sustainable Engineering and the Built Environment, Arizona State University, Tempe, AZ 85287-8101, United States of America
[3] Biodesign Center for Environmental Health Engineering, Biodesign Institute, Arizona State University, Tempe, AZ 85287-8101, United States of America
[4] Sioen Industries, Brussels, Belgium
[5] Department of Obstetrics and Gynecology, SUNY Downstate Health Sciences University, College of Medicine, Brooklyn, NY 11203, United States of America
[6] Authors to whom any correspondence should be addressed.

E-mail: Lori.Hoepner@downstate.edu, Rolf.Halden@asu.edu, Rolf.Halden@asu.edu, Benny.Pycke@sioen.be, Ovadia.Abulafia@downstate.edu, David.Sherer@downstate.edu and Laura.Geer@downstate.edu

Keywords: bisphenol A, prenatal, urine, maternal, urban

OPEN ACCESS

RECEIVED
22 September 2019

REVISED
10 March 2020

ACCEPTED FOR PUBLICATION
23 March 2020

PUBLISHED
6 April 2020

Abstract

Prior studies suggest ubiquitous fetal exposure to the endocrine disrupting chemical bisphenol A (BPA). Insufficient information is available on the effects of BPA in underserved urban populations in the US. We describe prenatal BPA exposures in a predominately Afro-Caribbean immigrant population. Maternal third-trimester urinary concentrations of total BPA were measured in 181 mothers in Brooklyn, NY from 2007 to 2009. Mothers aged 18–45 y presenting at a prenatal clinic consented to study participation. Spot urine samples were collected once between the sixth and ninth month of pregnancy. The geometric mean concentration of total BPA was 0.12 $\mu g\,l^{-1}$ (95% CI: 0.05–0.31). Total BPA concentrations were above the limit of detection in 9% of the mothers. Our results suggest that prenatal BPA exposure is low to non-detectable ($< 0.02\ \mu g\,l^{-1}$ in urine) among African American and Afro-Caribbean immigrant women residing in Brooklyn, NY. These results contradict evidence of prenatal exposure in prior studies of urban populations. Further studies should be conducted to determine whether there are associations between recent immigrant status and BPA exposures during pregnancy.

1. Introduction

Bisphenol A (BPA) is an endocrine disrupting chemical (EDC) commonly used as a monomeric building block and plasticizer in consumer plastic products such as polycarbonate plastics and epoxy resins. These common uses result in significant human exposure orally, dermally and via inhalation, with dietary intake representing the primary route [1–4]. Prenatal exposures may occur when conjugated BPA crosses the placenta *in utero* [5, 6]. Studies suggest associations between prenatal BPA exposures and adverse health outcomes in newborns and children [7–13].

To date, few studies have focused on prenatal BPA exposures in women of multiple races/ethnicities in the US [14–16]. Of these, only two studies recruited women from urban prenatal populations, both in NYC [14, 15]. Further, the two studies of urban prenatal populations are the only studies that included women of Latina-Caribbean descent from the Dominican Republic and Puerto Rico [14, 15]. However, no prior study in the US has assessed BPA exposures in pregnant women specifically of Afro-Caribbean descent. The present study aimed to assess urinary BPA concentrations and birth outcomes in a distinct prenatal NYC population of Afro-Caribbean women in Brooklyn.

2. Materials and methods

2.1. Study design and population

Subjects ($n = 181$) were a subset of women recruited for a study of prenatal mercury exposure with methods described previously [17]. Briefly, pregnant women were invited to participate in the larger study while attending the University Hospital of Brooklyn's Prenatal Clinic and recruited between October 2007 and December 2009. Criteria for study inclusion were singleton pregnancy, third trimester of pregnancy, and maternal age of 18 to 45 years. Maternal urine was collected once during the third trimester of pregnancy (mean gestational age: 33.26 weeks; SD: 1.79). A questionnaire was administered during enrollment to collect demographic data, medical history, mother's age, nativity, race/ethnic origin, and education level. Birth outcome data was abstracted from the delivery record. The study protocol was approved by the Institutional Review Boards of State University of New York Downstate Medical Center (SUNY DMC) and the New York State Department of Health. Study procedures and biological sample collection were explained to each subject at enrollment and a signed consent was obtained.

2.2. Urinary biomarker collection

Urine was collected as spot urine samples once during the sixth to ninth month of pregnancy. Samples were collected at varying times of the day. Total BPA urinary concentrations ($\mu g\,l^{-1}$) were measured at Arizona State University using offline solid phase extraction [18] followed by isotope dilution liquid chromatography tandem mass spectrometry as described earlier [19]. Specimens (1 ml of material urine) were thawed, spiked with a solution containing isotope-labeled standards (10 μl) as well as a solution containing two hydrolysis standards (50 μl), and diluted with a solution containing hydrolysis enzymes (1 mL). Analytes were extracted using a 24-port vacuum manifold and 60 mg of Oasis HLB (Waters, Milford, MA) solid-phase extraction cartridges. Aliquots (10 μl) of the resultant organic extracts were injected into a tandem mass spectrometer (API 4000 instrument; Applied Biosystems, Framingham, MA, USA), coupled to a Shimadzu Prominence HPLC (Shimadzu Scientific Instruments, Inc., Columbia, MD, USA). The assay's limit of detection (LOD) was 0.02 $\mu g\,l^{-1}$. For measurements below the LOD, a value equal to one half the LOD was assigned.

2.3. Creatinine determination

Urine aliquots of 2 ml were measured for creatinine onsite at SUNY DMC using the Alkaline Picrate Method and a Beckman Olympus Analyzer, model AU-2700 (Beckman Coulter, Inc., Brea, CA). Urinary creatinine adjustment accounts for dilution differences due to diurnal variation of random spot urine specimens collected at various times throughout the day.

2.4. Statistical analysis

Mean of the BPA concentrations was calculated, log-transformed, and exponentiated to calculate the geometric mean. Normality of the BPA concentration distribution was tested using the one-sample Kolmogorov-Smirnov test. Linear regression was used to determine associations between BPA concentration and birth outcomes. We considered results with $p<0.05$ to be statistically significant. Analyses were performed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA).

3. Results

Total BPA concentration ranged from <0.02 to 0.07 $\mu g\,l^{-1}$, with levels above the LOD detectable in urine from sixteen mothers or 9% of the maternal cohort. BPA concentrations had a geometric mean of 0.03 $\mu g\,l^{-1}$ (95% CI: 0.02–0.03). Creatinine values were available for thirteen of the sixteen subjects with BPA concentration above LOD (81%). Mean creatinine was 1.81 $g\,l^{-1}$ (SD: 1.02) for subjects with BPA values above LOD (n = 13) and 1.57 $g\,L^{-1}$ (SD: 0.80) for all subjects with creatinine (n = 169). Total BPA concentration was not normally distributed. Mean gestational age at time of urine collection was 33.26 w (SD: 1.79; range 26–37.5 w). Subjects with BPA concentration above LOD had a mean gestational age of 33.90 w (SD: 1.55; range 32–37 w) at the time of urine collection.

Demographic characteristics of the subjects are reported in table 1. Races/ethnicities were similarly represented across the age groupings. African American mothers were the majority race/ethnicity among those 24 y or younger, while Caribbean mothers were the majority race/ethnicity among 25–29 y and 30–34 y. Among those 35 y and older, the distribution was evenly split among African American and Caribbean mothers (n = 9, 37.5%). No African mothers were represented among the 30–34 y category, nor were Latina mothers represented among the 25–29 y category. Among mothers with detectable levels of BPA concentration, African American and Caribbean were evenly represented among age groups 24 y or younger and 30–34 y (n = 4,

Table 1. Socio-demographic characteristics of mothers.

Maternal characteristic	BPA > LOD (n = 16) n (%)	All subjects (n = 181) n (%)
Race/Ethnicity[b]		
African American	8 (50%)	77 (42.5%)
Caribbean	6 (37.5%)	75 (41.4%)
African	1 (6.3%)	6 (3.3%)
Latina	—	6 (3.3%)
Other	1 (6.3%)	14 (7.7%)
Age (y)		
⩽ 24	9 (56.3%)	68 (37.6%)
25–29	5 (31.3%)	46 (25.4%)
30–34	2 (12.5%)	42 (23.2%)
35+	—	25 (13.8%)
Marital status[2]		
Married	8 (50%)	46 (25.9%)
Not married	8 (50%)	131 (74.0%)
Health insurance[a, b]		
Public	15 (93.8%)	137 (75.7%)
US Born		
Yes	9 (56.3%)	95 (54.5%)
Education		
At least a HS diploma	15 (93.8%)	133 (73.5%)

[a] Missing data BPA > LOD: Health Insurance (n = 1);
[b] Missing data all subjects: Race/Ethnicity (n = 3), Marital Status (n = 4).

Table 2. Birth outcomes—all mothers (n = 181).

Birth outcomes (unit) (n)	Mean (SD)
Birth weight (g) (159)	3083.42 (564.96)
Birth length (cm) (154)	37.84 (2.16)
Head circumference (cm) (148)	49.42 (3.53)
Gestational age (w) (155)	37.86 (2.16)

44.4%; n = 1, 50% respectively). Only in the age group 25–29 y were African Americans of greater representation (n = 3, 60%). All race/ethnicities were represented across all education categories except Latina who either had less than a HS diploma (n = 2, 4.3%) or post-secondary school education (n = 4, 5.3%). Among those married, mothers of Caribbean descent were the majority race/ethnicity for all subjects (n = 46, 39.1%) and for those with BPA concentrations above detection (n = 5, 62.5%). In the full sample, 69.8% (n = 60) of mothers born outside the US were of Caribbean origin.

Birth outcomes are displayed in table 2. Data on birth outcomes were not available for all subjects. Of the 181 subjects, gestational age was available for 86% (n = 155) with 70% (n = 127) subjects who carried to full term. Of the pre-term births, 13% (n = 24) were pre-term, 2% were very pre-term (n = 3), and 1% (n = 1) were extremely pre-term. BPA concentration was above LOD for 11% of the 127 full term births (n = 14).

4. Discussion

Overall, Afro-Caribbean women of child-bearing age in Brooklyn do not have detectable nor elevated levels of BPA concentration as compared to other women in NYC and the US [14, 15, 20]. This study was conducted from 2007 to 2009 before bisphenol F and bisphenol S were widely used as substitutes for BPA in common consumer products. Thus, the fact that 91% of mothers had urinary BPA concentrations below the LOD cannot be attributed to the use of products made with alternative substances.

Given the small number of women with detectable concentrations of BPA (n = 16), comparative statistical analyses to the overall study population was not conducted. However, observation of the differences between women with BPA concentrations above LOD versus below LOD reveals that they are similar in the majority of characteristics except representation of Latina women, women aged 35 and older, and birth weight of offspring. Latina women comprise 3.3% (n = 6) of all subjects but none of the women with BPA concentrations above

LOD. Similarly, women aged 35 y or more were not among those with detectable levels of BPA concentrations. As noted above, the categories of Latina race/ethnicity and age 35 y or more were not exclusive. Thus, it is by expected probability based on the small number of Latina women who were enrolled in the study that no Latina mothers had BPA concentrations above LOD.

Of interest is the fact that no mothers aged 35 y or more had detectable concentrations of BPA. There is a general tapering off from youngest to oldest age group regarding proportion of those exposed to BPA. This gradual reduction may be an artifact of the overall age representation in the full study sample. However, as the consensus is that BPA exposure originates primarily from diet, it may be that older mothers have different feeding behaviors than younger mothers. For instance, older mothers may have lifestyles allowing them to be more able to avoid food storage and preparation materials that contain BPA or BPA epoxy resin as compared to younger mothers. In addition, diurnal variations in BPA concentrations throughout the day may account for the differences between older and younger mothers.

Urine was collected at earlier gestational ages among all mothers when compared to exposed mothers (ranges: 26–37.5 w versus 32–37 w, respectively). It is commonly accepted that BPA has an elimination time of approximately 4 h. However, while it is also known that BPA can cross the human placenta, there is no conclusive evidence regarding clearance rate of BPA from the developing human fetus. Given the variation in timing of urine collection and potential diurnal variations in urinary BPA concentrations, it is not possible to conclude that the fetuses were not also exposed to BPA.

It is widely accepted that most people in the US have been exposed to BPA based on NHANES results [1]. Furthermore, this study provides unexpected results in comparison to fifteen US-based prenatal studies conducted between 1999 and 2016, particularly those including African American and/or Caribbean origin mothers, that have BPA exposure among 62%–97% of the samples [21]. The aforementioned studies conducted analysis of urinary BPA concentrations. However, trimester of urine collection and laboratory methodology varied between studies. Additionally, while the other US-based studies sought to identify race/ethnicity, the foreign-born status was not reported. In this study, nearly half the mothers were born outside the US with the majority born in a Caribbean nation. Immigrant status may confer a protective effect due to cultural differences in dietary behaviors. The majority of mothers in the study received public health insurance which could be considered a proxy for low income status. The high proportion of non-exposure among majority low income mothers is consistent with prior research showing that income level or SES does not have a relationship with BPA exposure [14, 22].

Limitations of this study include the use of single spot urines and the lack of recording time of day for urine collection. Diurnal variations in urinary BPA concentrations have been reported and it is possible collection time may have affected our results. We also did not collect maternal dietary data, length of stay in the US, income level, or general health data. Strengths of this study include a diverse but understudied prenatal population residing in Brooklyn, NY. It is the only study conducted in NYC that has specifically aimed to characterize BPA exposures in Afro-Caribbean women.

5. Conclusions

Our results suggest that BPA may not be an exposure of concern among African American and Afro-Caribbean women residing in Brooklyn, NY. These results contradict evidence of prenatal exposure in prior US and NYC-based studies. This study provides evidence of differences in exposures to BPA which may be due to cultural differences in diet and dietary behaviors such as use of fresh food versus pre-packaged or ready-to-eat food. Culturally sensitive interventions may reduce exposures in diverse ethnic populations. In addition, further studies should be conducted to determine whether there are associations between recent immigrant status and BPA exposures during pregnancy.

Author contributions

Conceptualization, L A G and L A H; methodology, R U H and B P; software, L A H; formal analysis, L A H; resources, O A and D M S; data curation, L A G; writing - original draft preparation, L A H, L A G; writing - review and editing, R U H; visualization, L A H; supervision, O A and D M S; project administration, L A G; funding acquisition, R U H.

Funding

This research was funded by National Institute of Environmental Health Sciences, grant numbers R01ES015445, R01ES020889 and by the Virginia G Piper Charitable Trust, grant number LTR 05/01/12. The content is solely the responsibility of the authors and does not necessarily represent the official views of the funding agencies.

Conflicts of interest

The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

ORCID iDs

Lori A Hoepner https://orcid.org/0000-0002-4404-8140
Laura A Geer https://orcid.org/0000-0003-4300-9796

References

[1] Calafat A M, Ye X, Wong L, Reidy J A and Needham L L 2008 Exposure of the US population to bisphenol A and 4-tertiary-octylphenol: 2003-2004 *Environ. Health Perspect.* **116** 39–44
[2] Vandenberg L N, Hauser R, Marcus M, Olea N and Welshons W V 2007 Human exposure to bisphenol A (BPA) *Reprod. Toxicol.* **24** 139–77
[3] Vandenberg L N, Chahoud I, Heindel J J, Padmanabhan V, Paumgartten F J R and Schoenfelder G 2010 Urinary, circulating, and tissue biomonitoring studies indicate widespread exposure to bisphenol A *Environ. Health Perspect.* **118** 1055–70
[4] Wilson N K, Chuang J C, Lyu C, Menton R and Morgan M K 2003 Aggregate exposures of nine preschool children to persistent organic pollutants at day care and at home *J. Expo. Anal. Env. Epid.* **13** 187–202
[5] Ikezuki Y, Tsutsumi O, Takai Y, Kamei Y and Taketani Y 2002 Determination of bisphenol A concentrations in human biological fluids reveals significant early prenatal exposure *Hum. Reprod.* **17** 2839–41
[6] Schönfelder G, Wittfoht W, Hopp H, Talsness C E, Paul M and Chahoud I 2002 Parent bisphenol A accumulation in the human maternal-fetal-placental unit *Environ. Health Perspect.* **110** A703–7
[7] Rochester J R, Bolden A L and Kwiakowski cf 2018 Prenatal exposure to bisphenol A and hyperactivity in children: a systematic review and meta-analysis *Environ. Int.* **2018** 343–56
[8] Huo W *et al* Maternal urinary bisphenol A levels and infant lowbirth weight: a nested case-control study of the health baby cohort in China *Environ. Int.* 2015 **85** 96–103
[9] Whyatt R M, Rundle A G, Perzanowski M S, Just A C, Calafat A M, Hoepner L and Miller R L 2014 Prenatal phthalate and early childhood bisphenol A exposures increase asthma risk in inner-city children *J. Allergy Clin. Immun.* **134** 1195–7 e2
[10] Hoepner L A, Whyatt R M, Widen E M, Hassoun A, Oberfield S E, Mueller N T, Diaz D, Perera F P and Rundle A G 2016 Bisphenol A and adiposity in an inner city birth cohort *Environ. Health Perspect.* **124** 1644–50
[11] Ranjit N, Siefert K and Padmanabhan V 2010 Bisphenol-A and disparities in birth outcomes: a review and directions for future research *J. Perinatol.* **30** 2–9
[12] Roen E L, Wang Y, Calafat A M, Wang S, Margolis A, Herbstman J, Hoepner L A, Rauh V and Perera F P 2015 Bisphenol A exposure and behavioral problems among inner city children at 7-9 years of age *Environ. Res.* **142** 739–45
[13] Donohue K M *et al* Prenatal and postnatal bisphenol A exposure and asthma development among inner-city children *J. Allergy Clin. Immun.* 2013 **131** 736–42
[14] Hoepner L A, Whyatt R M, Just A C, Calafat A M, Perera F P and Rundle A G 2013 Urinary concentrations of bisphenol A in an urban minority birth cohort in New York City, prenatal through age 7 years *Environ. Res.* **122** 38–44
[15] Wolff M S, Engel S M, Berkowitz G S, Ye X, Silva M J, Zhu C, Wetmur J and Calafat A M 2008 Prenatal phenol and phthalate exposures and birth outcomes *Environ. Health Perspect.* **116** 1092–7
[16] Quirós-Alcalá L, Eskenazi B, Bradman A, Ye X, Calafat A M and Harley K 2013 Determinants of urinary bisphenol a concentrations in Mexican/Mexican-American pregnant women *Environ. Int.* **59** 152–60
[17] Geer L A, Persad M D, Palmer C D, Steuerwald A J, Dalloul M, Abulafia O and Parsons P J 2012 Assessment of prenatal mercury exposure in a predominately Caribbean immigrant community in Brooklyn, NY *J. Environ. Monit.* 1035–43
[18] Geer L A, Pycke B F G, Waxenbaum J, Sherer D M, Abulafia O and Halden R U 2017 Association of birth outcomes with fetal exposure to parabens, tricloan and triclocarben in an immigrant population in Brooklyn, New York *J. Hazard. Mater.* **323A** 177–83
[19] Minguez-Alarcon L *et al* The earth study team. Urinary concentrations of bisphenol A, parabens and phthalate metabolite mixtures in relation to reproductive success among women undergoing *in vitro* fertilization *Environ. Int.* 2019 **126** 355–62
[20] Woodruff T J, Zota A R and Schwartz J M 2011 Environmental chemicals in pregnant women in the United States, NHANES 2003-2004 *Environ. Health Perspect.* **119** 878–85
[21] Hoepner L A 2019 Bisphenol a: a narrative review of prenatal exposure effects on adipogenesis and childhood obesity via peroxisome proliferator-activated receptor gamma *Environ. Res.* **173** 54–68
[22] Hefferman A L, Sly P D, Toms L M L, Hobson P and Mueller J F 2014 Bisphenol a exposure is not associated with area-level socioeconomic index in Australian children using pooled urine samples *Environ. Sci. Pollut. R.* **21** 9344–55