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Assessing inter-generational transfer of a brominated flame retardant

Avenel D. Joseph,[a] Metrecia L. Terrell,[b] Chanley M. Small,[b] Lorraine L. Cameron[c] and Michele Marcus*[bd]

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Studies have shown that the lipophilic nature of polybrominated biphenyl (PBB) causes it to preferentially accumulate in breast milk posing a potential hazard for suckling infants. The purpose of this study was to examine the inter-generational transfer of PBB from mother to child and whether this association was modified by maternal breast-feeding patterns. One hundred and forty-five mother–child pairs that were participants of the Michigan Long-Term PBB Study were included in this analysis. Mothers were exposed to PBB *via* contaminated food between 1973 and 1974 and children were exposed *in utero* and for some, through breast-feeding. Seventy-three percent of children had a non-detectable serum PBB concentration (limit of detection (LOD) = 1 μg L^{-1}). Mothers' serum PBB concentration at enrollment ranged from <LOD to 933 μg L^{-1}. The following variables were associated with the child having a detectable serum PBB concentration: maternal serum PBB ≥8 μg L^{-1}, breast-feeding ≥5.5 months, maternal age at child's birth ≥28 years, and being born during the PBB exposure period. Among mothers with a detectable serum PBB concentration, those who breast-fed ≥5.5 months were 6 times more likely to have a child with a detectable serum PBB concentration, compared to a non-breast-fed child (95% C.I., 2.0–19.6).

Introduction

Extensive research on the composition of maternal milk shows that it contains an optimal balance of carbohydrates, fats, proteins, vitamins and hormones. In addition, breast milk contains potent immunological factors that protect against infectious and non-infectious diseases[1] and may also decrease the risk of chronic diseases such as diabetes and obesity.[2,3] Furthermore, the act of breast-feeding builds a bond between mother and child that is recognized to have lasting physiological and psychological benefits.

The presence of environmental toxicants poses a potential risk of breast-feeding to the suckling infant.[4] Contamination of breast milk with toxicants has been acknowledged since the 1950's[5] and has been increasingly gaining attention in recent years.[6] Studies in humans and animals have suggested that lipophilic toxicants are particularly hazardous to offspring because of their preference to partition into lipid rich deposits, such as breast milk. Brominated flame retardants (BFRs) are a group of lipophilic compounds that have been widely used as a chemical flame retardant in electronics and industrial products. A particular BFR, polybrominated biphenyl (PBB) has been found to be over 100-fold more concentrated in breast milk when compared to maternal serum.[7,8] In addition, there is evidence that a structurally related class of compounds, polybrominated diphenyl ethers (PBDEs), have been increasing in concentration within breast milk over the past two decades despite the fact that the manufacture of several of the more persistent PBDE congeners has ceased.[9] The potential for the lactational transfer of these chemicals may be particularly dangerous for a newborn undergoing rapid growth and development of the endocrine and nervous systems.

The widespread production and use of BFRs coupled with their environmental persistence and bioaccumulation raises concern about their potential health effects.[9–11] In humans, PBB may act as a potential endocrine disruptor in women.[12,13] There is also evidence that PBB can be transferred during pregnancy to offspring.[8,14,15]

Between 1973 and 1974 an industrial incident that resulted in Michigan animal feed being inadvertently contaminated with PBB, caused widespread contamination of the Michigan food supply. In the months following the accident, many Michigan residents unknowingly consumed meat, eggs and dairy products that were contaminated with PBB. As a result of this incident and subsequent pubic health concerns regarding adverse health effects from PBB exposure, the Michigan Department of Community Health (MDCH) established a registry of exposed Michigan residents (named the Michigan Long-Term PBB Study) in 1976. Further details of the incident and the cohort have been described previously.[14,16–18]

Several breast milk-monitoring studies have been conducted among Michigan PBB residents. Brilliant *et al.* reported that 73% of breast milk samples from a random sample of ninety-five Michigan nursing mothers had detectable PBB in breast milk fat (median = 68 micrograms per liter (μg L^{-1})).[19] Miller *et al.* reported similar breast milk levels (median = 60 μg L^{-1}) from 2900 Michigan nursing mothers who provided voluntary breast milk specimens to MDCH during 1976–1978.[20] A panel of experts that assembled to form recommendations for breast-feeding

[a]*Emory University, School of Medicine, Department of Cell Biology, Atlanta, Georgia, USA*
[b]*Department of Epidemiology, Rollins School of Public Health, Emory University, 1518 Clifton Rd NE, Atlanta, Georgia, 30322, USA. E-mail: mmarcus@sph.emory.edu; Fax: +404-727-8737; Tel: +404-727-8010*
[c]*Division of Environmental Health, Michigan Department of Community Health, Lansing, Michigan, USA*
[d]*Department of Environmental and Occupational Health, Rollins School of Public Health, Emory University, Atlanta, Georgia, USA*

mothers during that time period concluded that the benefits of breast-feeding far outweighed the potential risks.[21] However, a retrospective examination of breast-feeding patterns within the Michigan Long-Term PBB Study suggested that women who were in the highest PBB exposure group were less likely to breast-feed their infants in the first five years following the PBB exposure incident, although the trend was not significant.[22,23] Two studies have previously reported serum PBB concentrations among children in the long-term PBB study. Jacobson *et al.* reported the PBB concentration of children when they were 4 years old (min–max: not detectable–9.5 μg L^{-1}) and found that all of the children with detectable serum PBB had been breast-fed for at least 4 months.[24] Anderson and Wolff found a positive relationship between longer breast-feeding duration and higher serum levels of PBB in children.[25] Neither study compared the child's serum PBB concentrations to the mother's serum PBB concentrations.

The current study examines whether maternal serum PBB concentration is related to their child's serum PBB concentration and whether this association is modified by maternal breast-feeding patterns. The mothers were exposed to PBB through diet. The children were born after the contamination episode began and were exposed to PBB mainly *in utero* and for some, through breast-feeding.

Methods

Study population

The Michigan Long-Term PBB Study originally enrolled families that lived on Michigan farms or families that consumed food from quarantined farms. Since 1978, expanded enrollment periods were established to enroll children born to exposed mothers already enrolled in the cohort. To date, the cohort includes approximately 4000 active first, second, and now third generation individuals. Many of the first-generation women have had blood collected and analyzed at enrollment in the PBB cohort (primarily between 1976–1979) and some at multiple times during 1976–1993; however, only a small subset of the second-generation have ever had their blood collected and subsequently analyzed for PBB.

The study population was composed of second-generation children who have had a serum sample collected and analyzed for PBB ($n = 189$). We were able to identify the mothers of 181 children from cohort files. Two additional mother–child pairs were excluded because the mother did not have a serum sample collected. Information on demographics, socioeconomic indicators, lifestyle questions including smoking status, a detailed reproductive history, breast-feeding history and duration of lactation was obtained from maternal telephone interviews completed during 1997–1998 and 2003–2006. After removing mothers that did not participate in either telephone interview (for whom we do not have breast-feeding information), the final sample size was 145 mother–child pairs. The sample includes up to four siblings per family and includes three twin sets.

Exposure assessment

The PBB mixture that entered the Michigan food chain was primarily FireMaster FF-1. The main congener in FireMaster FF-1 (approximately 60% of the total mixture) is 2,2′4,4′5,5′–hexabromobiphenyl, also known as PBB–153. Serum was analyzed for PBB by the MDCH Bureau of Laboratories using gas chromatography with electron capture detection. Serum samples were denatured, extracted with ether–ethyl or hexane–ether, and passed through a column of Florisil or Florisil–silica gel.[26,27] A known quantity of FireMaster FF-1 was used as the calibration standard and sample peak sizes were compared to standard peak sizes. The coefficient of variation, determined from *in vivo* pools of bovine serum analyzed repeatedly for PBB concentrations ranging from 10 μg L^{-1}–100 μg L^{-1} were 7.1–14.0% and recovery ranges were 80–90%.[26] The limit of detection (LOD) for the PBB samples was 1.0 μg L^{-1}. PBB samples were collected from non-fasting participants and lipids were not measured.

In addition to maternal serum PBB concentration at enrollment into the cohort, we examined the estimated maternal PBB at the time of conception of the index child. This estimate was derived from a mixed-effects PBB decay model that accounts for time since the mother's PBB exposure, along with maternal factors of age, body mass index (BMI), smoking history, parity and breast-feeding history.[28]

Statistical analysis

To visually explore the relationship between maternal breast-feeding status and child's serum PBB concentration, we created a scatter plot of mother's and child's serum PBB concentrations stratified by breast-feeding status. Serum PBB concentrations below the LOD were set to half the LOD.[29] All concentrations were log-transformed because they were highly skewed. Separate linear regression lines were added to the scatter plot to represent child's breast-feeding status (no/yes).

In all other analyses, the outcome variable, child's serum PBB concentration, was dichotomized into child's PBB <LOD and child's PBB ≥LOD because 73% ($n = 106$) of the children did not have a detectable serum PBB concentration (<1 μg L^{-1}). For the mothers' serum PBB concentrations, 21% were undetectable and maternal PBB concentrations were categorized as <LOD, LOD up to the 75th percentile of 8 μg L^{-1} and ≥8 μg L^{-1}. Several maternal characteristics were considered as potential confounding variables: age at the child's birth, smoking the year before the index pregnancy, BMI at enrollment, parity since PBB exposure, and breast-feeding history since PBB exposure. Descriptive statistics and correlations between variables were evaluated. In bivariate analyses, each variable was modeled with the outcome, child's PBB level, and then with the main exposure of interest, child's breast-feeding status. To account for the lack of independence among siblings, bivariate and multivariate analyses were performed with generalized estimating equations (GEE). All analyses utilized SAS statistical software v9.1.[30]

In determining whether there was an association between serum PBB concentrations in children and their PBB exposed mothers, we restricted our multivariate analyses to include only mothers who had a detectable enrollment serum PBB concentration ($n = 116$ children). When estimating the serum PBB concentrations at conception, twelve children were excluded because their mothers estimated PBB concentration fell below the LOD. We expected that mothers with undetectable serum

PBB concentrations would transfer little, if any PBB *in utero* or through breast-feeding to their children. A stratified analysis confirmed this except in one mother–child pair where the mother had an undetectable serum PBB concentration and the child had a detectable serum PBB concentration. This child was born soon after the start of the PBB incident, was not breast-fed, and had a serum PBB concentration at the limit of detection (1 μg L^{-1}). The source of the child's exposure was likely due to consumption of contaminated dairy products rather than placental transfer or post-natal transfer through breast-feeding. In all, twenty-eight mother–child pairs were excluded because the mother had an undetectable serum PBB concentration. The remaining 116 mother–child pairs were included in the GEE models. Multivariate models including all potential confounding variables (maternal age, smoking history, BMI at enrollment, parity, prior breast-feeding history, time since exposure and child's age at serum PBB measurement) and the main exposure of interest, child's breast-feeding status, were considered. We then eliminated potential confounders if their elimination did not change the odds ratio (OR) of child's breast-feeding status by at least 10%. The maternal PBB concentration (enrollment or estimated at conception) was retained regardless. We computed Wald Chi-Square *p*–values and confidence intervals (C.I.) as warranted. We used an interaction term to examine the combined effect of child's breast-feeding status and maternal PBB concentrations on the probability of the child having a detectable serum PBB.

A small subset of children ($n = 20$) were born during the PBB exposure period and could have had access to PBB through dietary means other than *in utero* or through breast milk. Because of this, we also conducted analyses stratified by whether the child was born during or after the PBB exposure period. Finally, our sample included three twin sets, none of which were breast-fed. We randomly excluded one child from each twin set. Model estimates were not altered by the removal of three children. Therefore, we present our results including all eligible mother–child pairs including twins.

Results

The PBB exposure period lasted from July 1973 to May 1974, when the farms were quarantined. The mothers ($n = 112$) were exposed to PBB when they were 10 to 42 years of age (median maternal age at exposure = 21 years). Their serum PBB concentrations were determined at enrollment into the long-term cohort during 1976–1979 and ranged from below the LOD to 933 μg L^{-1} (median = 2 μg L^{-1}). The children ($n = 145$) were born from 1973–1982 and had their serum analyzed for PBB during infancy to 17 years of age (child's median age at serum PBB measurement = 4 years). As expected, the children had serum PBB concentrations that were substantially lower than their mothers. There were 39 children that had serum PBB concentrations at or above the LOD (min–max: 1–482 μg L^{-1}, median = 2.9 μg L^{-1}). Of these 39 children, 62% ($n = 24$) were breast-fed and of these 67% ($n = 16$) were breast-fed for 5.5 months or longer (Table 1). For the remaining 106 children that had serum PBB levels below the LOD, 43% ($n = 46$) were breast-fed and of these 43% ($n = 20$) were breast-fed for 5.5 months or longer (Table 1). The highest exposed mothers in our sample had PBB concentrations ≥100 μg L^{-1} ($n = 5$ mothers). Of their nine children, only the two that were breast-fed had PBB concentrations that were ≥100 μg L^{-1}.

Fig. 1 is a scatterplot and regression of child's PBB concentration in relation to mother's concentration stratified by breast-feeding status. The slope of the regression for breast-fed children

Table 1 Study population characteristics of mothers in the Michigan Long-Term PBB Study and their children, n=145 children, 112 mothers[a]

	Child's PBB concentration		
Characteristic	<LOD (106) *n* (row %)	≥LOD (39) *n* (row %)	Unadjusted OR[b]
Breast-feeding duration, months			
Not breast-fed	60 (80.00)	15 (20.00)	1.00
0.5–5.5	26 (76.47)	8 (23.53)	0.78 (0.26–2.34)
≥5.5	20 (55.56)	16 (44.44)	2.21 (0.97–5.02)
Mother's enrollment PBB, μg L^{-1}			
<LOD	28 (96.55)	1 (3.45)	1.00
1.0 – <8.0	66 (79.52)	17 (20.48)	7.15 (0.90–56.95)
≥8.0	12 (36.36)	21 (63.64)	41.60 (4.82–359.11)
Mother's estimated PBB at conception, μg L^{-1}			
<LOD	39 (95.12)	2 (4.88)	1.00
1.0 – <8.0	55 (77.46)	16 (22.54)	5.63 (1.24–25.55)
≥8.0	12 (36.36)	21 (63.64)	28.68 (5.68–144.76)
Time from mother's exposure to child's birth, months			
0–24	30 (63.83)	17 (36.17)	1.00
25–81	32 (71.11)	13 (28.89)	0.91 (0.34–2.43)
82–108	44 (83.02)	9 (16.98)	0.64 (0.21–1.93)
Child's age at assay, years			
1–3	29 (64.44)	16 (35.56)	1.00
4–7	38 (73.08)	14 (26.92)	0.28 (0.10–0.76)
8–17	39 (81.25)	9 (18.75)	0.26 (0.10–0.70)
Mother's age at child's birth, years			
16–22	35 (87.50)	5 (12.50)	1.00
23–27	38 (66.67)	19 (33.33)	2.55 (0.79–8.22)
28–43	33 (68.75)	15 (31.25)	3.43 (1.02–11.54)
Mother's BMI at enrollment, kg m^{-2}			
<18.5	6 (66.67)	3 (33.33)	1.41 (0.31–6.39)
18.5–24.9	65 (69.89)	28 (30.11)	1.00
25.0–29.9	22 (91.67)	2 (8.33)	0.25 (0.05–1.15)
≥30	13 (68.42)	6 (31.58)	0.91 (0.26–3.19)
Smoking, year before pregnancy			
No	86 (71.07)	35 (28.93)	1.00
Yes	20 (83.33)	4 (16.67)	0.67 (0.23–1.90)
Sex of child			
Male	58 (78.38)	16 (21.62)	1.0
Female	48 (67.61)	23 (32.39)	1.35 (0.65–2.81)
Parity since exposure, at index child			
0	66 (70.97)	27 (29.03)	1.00
1	31 (75.61)	10 (24.39)	1.15 (0.51–2.57)
≥2	9 (81.82)	2 (18.18)	0.81 (0.43–1.50)
Child's birth in relation to exposure			
After exposure (6/1974 – 7/1982)	96 (78.05)	27 (21.95)	1.00
During exposure (7/1973 – 5/1974)	10 (45.45)	12 (54.55)	2.29 (0.72–7.28)

[a] Abbreviations: LOD, limit of detection; μg L^{-1}, micrograms per liter. [b] Unadjusted ORs are the odds of a child having a detectable serum PBB concentration.

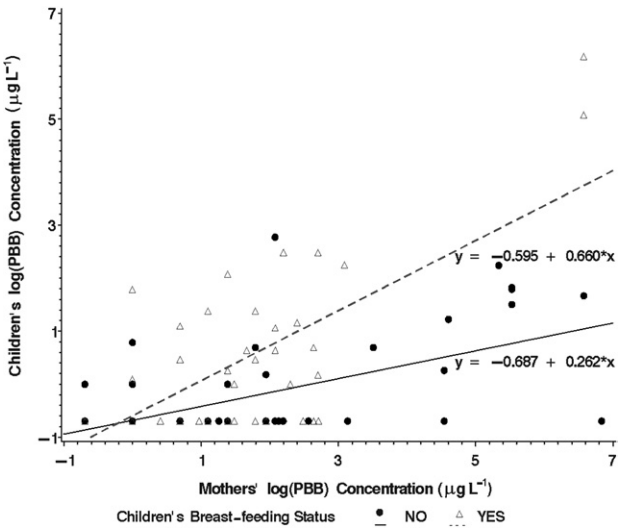

Fig. 1 Relationship between mothers' serum PBB concentrations and their children's serum PBB concentrations modified by breastfeeding ($n =$ 145 children). Log represents natural logarithm. Undetectable PBB concentrations set to (Limit of Detection (LOD)/2).

was significantly steeper than for non-breast-fed children. In addition to breast-feeding, several other covariates were associated with the child having a detectable serum PBB concentration (Table 1): maternal serum PBB ≥8 μg L^{-1}, maternal age at child's birth ≥28 years, and being born during the PBB exposure period.

In Table 2, we present a multivariate model including breast-feeding duration, maternal PBB concentration (enrollment or estimated at the child's birth), and an indicator variable for whether the child was born during the PBB exposure period. Maternal age was related to the mother's decision to breast-feed, but was not related to maternal serum PBB level. As a result, maternal age was not retained in the model because it was not a confounder. Among mothers with a detectable serum PBB concentration, those who breast-fed for ≥5.5 months were 6 times more likely to have a child with a detectable serum PBB concentration when compared to a child who was not breast-fed (OR = 6.2; 95% C.I., 2.0–19.6), adjusted for child's birth in relation to exposure period. A history of prior breast-feeding since exposure by the mother attenuated the effect of breast-feeding duration slightly (OR = 5.46; 95% C.I., 1.7–17.8), but was not a confounder in the models. Mothers with a serum PBB concentration ≥8 μg L^{-1} were 11 times more likely to have a child with a serum PBB concentration above the LOD (OR = 11.1; 95% C.I., 3.7–33.6). The odds ratios were similar when using the maternal estimated PBB at conception.

Table 3 gives the interaction model for the effect of breast-feeding status (no/yes) and maternal serum PBB (1.0 – <8.0, μg L^{-1}). Mother's age at child's birth and child's birth in relation to exposure did not confound the relationship between breast-feeding and child's detectable PBB, thus we present the unadjusted model. Mothers who had a serum PBB concentration ≥8 μg L^{-1} and who breast-fed were the most likely to have a child with a detectable serum PBB concentration (OR = 26, 95% C.I., 5.2–130.7).

Table 3 Unadjusted interaction model of ORs and 95% C.I.s for the odds of a child having a detectable serum PBB concentration when born to an exposed mother [ab]

Effect				
Child's breast-feeding status	Maternal enrollment PBB concentration (μg L^{-1})	*n*	OR	95% C.I.
No	1.0 – <8.0	44	1.00	—
Yes	1.0 – <8.0	39	3.24	1.06, 9.96
No	≥8.0	18	6.95	1.78, 27.16
Yes	≥8.0	15	26.04	5.19, 130.66

[a] Exposed mother defined as having a detectable enrollment serum PBB concentration. [b] Abbreviations: OR, odds ratio; C.I., confidence interval; μg L^{-1}, micrograms per liter.

Table 2 Adjusted ORs and 95% C.I.s for the odds of a child having a detectable serum PBB concentration when born to an exposed mother[ab]

	Maternal			Maternal		
	Enrollment PBB			Estimated PBB at conception		
Effect	*n*	AOR[c]	95% C.I.	*n*[d]	AOR[c]	95% C.I.
Breast-feeding duration, months						
Not breast-fed	62	1.00	—	57	1.00	—
0.5–5.5	24	2.05	0.61, 6.97	21	2.05	0.59, 7.13
≥5.5	30	6.18	1.95, 19.60	26	5.73	1.78, 18.47
Maternal PBB concentration[e], μg L^{-1}						
1.0 – <8.0	83	1.00	—	71	1.00	—
≥8.0	33	11.14	3.69, 33.62	33	9.36	3.10, 28.23
Child's birth in relation to exposure						
After exposure (6/1974 – 7/1982)	96	1.00	—	85	1.00	—
During exposure (7/1973 – 5/1974)	20	6.04	1.84, 19.82	19	5.66	1.69, 19.02

[a] Exposed mother defined as having a detectable enrollment/estimated serum PBB concentration. [b] Abbreviations: AOR, adjusted odds ratio; C.I., confidence interval; μg L^{-1}, micrograms per liter. [c] Adjusted only for the effects given in the table. [d] Twelve children were excluded from the estimated model because maternal estimated PBB <LOD. [e] PBB represents maternal serum PBB concentrations at the time of enrollment or at conception.

Discussion

This article examines the inter-generational transfer of a particular brominated flame retardant, PBB, from mothers to children in the Michigan Long-Term PBB study. In the present study, mother's serum PBB concentration was the most important predictor of her child having a detectable serum PBB concentration. We found that the mother's breast-feeding status (no/yes) and breast-feeding duration in months were the next strongest predictors. Particularly, mothers who breast-fed their children for ≥5.5 months increased the chance that their child would have a detectable blood concentration of PBB. In addition, the association between maternal and child serum PBB concentrations was stronger for those born during the exposure period than for those born after the exposure period.

We investigated several maternal characteristics and their relationship to whether an offspring had a detectable serum PBB concentration. The socioeconomic factors of maternal education or income were not considered as confounding variables because they were captured at the study interviews, which occurred several years after the children were born and were not a reliable proxy for maternal socioeconomic status at the time of the offspring's birth. Another study that considered socioeconomic status found that it was not a significant predictor of child's serum PBB concentrations, when adjusted for breast-feeding length.[24] Similarly, maternal BMI was assessed at enrollment into the cohort and at the study interviews and was not available at an appropriate time for each pregnancy.

Because maternal breast-feeding information was obtained retrospectively, we were concerned about the accuracy of reporting for breast-feeding duration, and the effect this would have on our conclusions. However, when we classified breast-feeding by decision (no/yes) we still observed a significant association with the probability of a child's detectable serum PBB (OR = 3.8; 95% C.I., 1.4–10.1). This measure is less likely to be subject to recall inaccuracies, particularly because we previously found very little association between maternal serum PBB and decision to breast feed (no/yes).[22] In our sample, breast-feeding status did not differ by mother's serum PBB concentration.

PBB measurements of the children in this study were not taken when they were the same age. Therefore, it is possible that we may have underestimated our results for children who had PBB measured as teenagers because the elimination of PBB may be different in children, due to variations in growth and metabolism at different stages of development. When we added child's age at serum PBB measurement to the model in Table 2, we found that children in our oldest age group (8–17 years) were less likely to have a detectable serum PBB when compared to children 1–3 years old at the PBB measurement (OR = 0.09; 95% C.I., 0.02–0.5). Thus, if PBB measurements were taken closer to when the children were breast-fed, it is likely that more children would have had a detectable serum PBB concentration.

In contrast to the few studies of PBBs, the lactational transfer of a closely related group of compounds known as polychlorinated biphenyls (PCBs) have been well documented in humans. The transmission of PCB in breast milk was reported in 1980 when a study of Japanese women and their offspring revealed that PCB concentrations increased at 3 months postpartum in breast-fed infants compared to bottle-fed infants (2.4 μg L^{-1} *vs* 0.6 μg L^{-1}, respectively).[31] In 1984, an independent group confirmed these results and reported that non-breast-fed children had a mean serum PCB of 4.6 μg L^{-1} *vs* 42.4 μg L^{-1} for children breast-fed ≥3 months.[32] It has been estimated that each week of breast-feeding increases the offspring's plasma concentration by 0.3% of the mother's PCB concentration in her breast milk.[33]

We were unable to examine PBB concentrations in maternal breast milk due to the fact that very few breast milk samples were available. However, for a small subset of participating mothers (n = 12), breast milk levels ranged from 30 to 92 667 μg kg^{-1} lipid and the median was 216 μg kg^{-1} lipid. Maternal breast milk levels of PBDEs measured in two U.S. populations have been much lower with medians ranging from 19 to 34 μg kg^{-1} lipid.[34,35] However, given the similar behavior of this class of chemicals, the transfer of PBDEs to a suckling infant is still likely.

In our study, when we compared the maternal milk and serum concentrations of PBB for the 12 mothers, the results indicated that the maternal milk : serum PBB ratio was approximately 100 : 1. These results are consistent with a previous report that found a similar maternal milk : serum PBB ratio of 107–119 : 1, in 46 women.[7]

One of the major strengths of this study is that we have serum measurements of both maternal and offspring PBB concentrations. This is one of only a few reports that have investigated the relationship between offspring serum PBB concentration and mother's breast-feeding history. In the other two studies, PBB was measured when the children were between the ages of 4 and 5[24] or PBB was measured in 1978 from children of varying ages, which likely included first-generation children born before the PBB exposure incident.[36] In comparison, our study included second-generation children whose primary routes of PBB exposure would have been *in utero* or through breast-feeding and who had serum PBB collected at various childhood ages. However, we cannot entirely rule out some dietary exposure even after the farms were quarantined and all contaminated products were supposed to be destroyed.

Conclusion

This article shows that the inter-generational transfer of PBB is similar to that of other related lipophilic compounds such as PCB and PBDE. Future studies will examine the potential health effects in children exposed to PBB *in utero* and through breast-feeding. To date, this is the only study for PBB that has shown a relationship between maternal and child serum concentrations and how this relationship is accentuated by breast-feeding.

Abbreviations

BMI	body mass index
μg L^{-1}	micrograms per liter
GEE	generalized estimating equations
μg kg^{-1}	micrograms per kilogram
BFR	brominated flame retardant
PBB	polybrominated biphenyl
PCB	polychlorinated biphenyl
PBDE	polybrominated diphenyl ethers (PBDEs)

MDCH	Michigan Department of Community Health
LOD	limit of detection
OR	odds ratio
C.I.	confidence interval

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