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Authors

Lancz, Kinga
Hertz-Picciotto, Irva
Jusko, Todd A
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Duration of breastfeeding and serum PCB 153 concentrations in children

Kinga Lancz[1], **Irva Hertz-Picciotto**[2], **Todd A. Jusko**[3], **Ľubica Murínová**[1], **Soňa Wimmerová**[1], **Eva Šovčíková**[1], **Ladislav Dedík**[4], **Maximilián Strémy**[5], **Beata Drobná**[1], **Dana Farkašová**[1], and **Tomáš Trnovec**[1]

Kinga Lancz: kinga.lancz@szu.sk; Irva Hertz-Picciotto: ihp@ucdavis.edu; Todd A. Jusko: todd_jusko@urmc.rochester.edu; Ľubica Murínová: lubica.palkovicova@szu.sk; Soňa Wimmerová: sona.wimmerova@szu.sk; Eva Šovčíková: eva.sovcikova@szu.sk; Ladislav Dedík: ladislav.dedik@stuba.sk; Maximilián Strémy: maximilian.stremy@stuba.sk; Beata Drobná: beata.drobna@szu.sk; Dana Farkašová: rektor@szu.sk; Tomáš Trnovec: tomas.trnovec@szu.sk

[1]Slovak Medical University, Limbová 14, 83303 Bratislava, Slovakia

[2]Division of Environmental and Occupational Health, Department of Public Health Sciences, University of California, Davis CA 95616

[3]Division of Epidemiology, Department of Public Health Sciences, University of Rochester School of Medicine and Dentistry, Rochester, NY 14642

[4]Slovak University of Technology in Bratislava, Faculty of Mechanical Engineering, Vazovova 5, 812 43 Bratislava 1, Slovakia

[5]Research Centre of Progressive Technologies, Faculty of Materials Science and Technology in Trnava, Slovak University of Technology in Bratislava, Hajdóczyho 1, 917 24 Trnava, Slovakia

Abstract

Polychlorinated biphenyls (PCBs) are toxic, persistent, and bioaccumulative chemicals which, because of their lipophilic properties, are abundant in human breast milk. Breastfed infants are therefore at risk of being exposed to considerable amounts of PCBs. The commonly used exposure estimations, based solely on breast milk PCB levels and duration of breastfeeding, may lead to exposure misclassification. To improve assessments of exposure to PCBs, we determined PCB 153 serum concentration, as a model substance for PCBs, at the critical time of weaning for each child in 305 breastfed infants from 5 single time point concentration measurements spread over 7 years and data on duration of breastfeeding, using an earlier developed model of the system type. We approximated the dependence of PCB 153 serum concentration, C_{tbf}, adjusted to cord serum concentration, C_0, on nursing period, by a polynomial function $C_{tbf}/C_0=0.596+0.278t-0.0047t^2$ which reliably predicts exposure to PCB 153 of breastfed infants, important for assessment of

Corresponding author, Dr. Tomáš Trnovec, Slovak Medical University, Limbová 12, 83303 Bratislava, Slovakia, tomas.trnovec@szu.sk. Telephone: +4212 59370225, Fax: +4212 59370151.

dose-outcome relationships. Adjustment of current serum concentrations to cord serum concentration improved validity of exposure assessment.

Keywords

breastfeeding; PCB exposure assessment; PCB 153 serum concentration; polynomial function; toxicokinetics

1. Introduction

Large quantities of toxic polychlorinated biphenyls (PCBs) have been released into the environment as a result of PCB production and widespread use. Because of their high lipophilicity, PCBs bioaccumulate in the fatty tissues of organisms. For example, human milk with its 3–5 % fat content (Jenness, 1979) is an important carrier of these compounds and lactational transfer represents one of the principal routes of PCB exposure to developing mammals, including humans. The concentration time course of organochlorines in blood during nursing is peaking at the time of weaning and therefore many authors consider duration of breastfeeding a measure of exposure. Higher perinatal exposures to organochlorines, such as PCBs, have been associated with poorer cognitive development (Shonkoff et al., 2009; Shonkoff and Garner, 2012) and deficits of immune system function (Grandjean et al., 2008). Therefore assessment of accompanying toxicokinetic processes is essential for understanding relevant dose-effect relationships.

A measure of exposure of infants to lipophilic organochlorines through breastfeeding is the amount of milk taken up into the body. Therefore the duration of breastfeeding is a strong predictor of body burden of organochlorines (Hardell et al., 2010; Lackmann et al., 2004; Patandin et al., 1997; Patandin et al., 1999; Kreuzer et al., 1997; Barr et al., 2003; Lanting et al., 1998; Chao et al., 2004; Tohyama et al., 2011). A more accurate assessment of exposure is a product of the concentration of the compound in milk and the rate of consumption of breast milk (Patandin et al., 1999; Bergkvist et al., 2010; Arcus-Arth et al., 2005; Koopman-Esseboom et al., 1996; Ulaszewska et al., 2011) or alternatively a product of the breast milk levels and the number of weeks of breastfeeding (Pan et al., 2009; Pan et al., 2010; Weisglas-Kuperus et al., 2004). All these approaches are accompanied by many uncertainties. Known is imprecision of reporting duration of breastfeeding by the mother and also data on exclusive breastfeeding may imprecisely reflect the situation (Aarts et al., 2000). We have recently shown that estimations based solely on breast milk PCB levels and duration of breastfeeding may lead to exposure misclassification (Verner et al., 2013).

In 2002–2003 we started long term observation of children living in an area polluted by PCBs and information on lactational exposure to PCBs of each child of our cohort was essential for description of the respective concentration-effect relationships (Hertz-Picciotto et al., 2003). We had two options. The first was to apply a system model (Trnovec et al., 2011) using data on time series serum concentration measurements and duration of breastfeeding. The second was based on PBPK simulations using individual physiologic parameters, duration of breastfeeding, and levels of PCBs measured in maternal blood at delivery, cord blood, or breast milk (Verner et al., 2013). In the currently used former option

we describe dependence of the concentration of PCB 153 in serum of 305 breastfed infants on length of breastfeeding. Although we determined the concentrations of 15 PCB congeners in serum specimens, we focused our analyses on PCB-153 for two reasons: 1) PCB-153 is highly correlated with total PCB concentration in this cohort (Jusko et al., 2010), and 2) it was detectable in the vast majority of child specimens (Kocan et al., 2004).

2. Material and methods

We included into the study altogether 305 infants from Michalovce district in eastern Slovakia (Hertz-Picciotto et al., 2003). This is a subgroup from originally 1082 children for which data for all 5 measurement times (cord blood at birth, 6, 16, 45 and 72 months later) were available. We have shown that for a slightly different subgroup maternal PCB-153 concentrations were highly similar between participants in the subgroup study and the entire cohort at birth (p=0.99) (Jusko et al., 2014). The Michalovce area was polluted by PCBs which were produced at the chemical facility Chemko Strážske. The extent of pollution can be illustrated by comparing the median serum concentration for the same time period in 1009 adult subjects for congener PCB 153 of 578 ng/g of lipid in the Michalovce district (Petrik et al., 2006) with the median serum concentration in 415 nonpregnant women for congener PCB 153 of 8.3 ng/g of lipid in USA (Woodruff et al., 2011). The characteristics of infants and mothers participating in the study were described earlier (Jusko et al., 2012; Jusko et al., 2011; Jusko et al., 2010; Park et al., 2010; Sonneborn et al., 2008a, 2008b). The study protocol was approved by Institutional Review Boards at the University of California, Davis and the Slovak Medical University. Mothers gave informed consent and were enrolled at the time they came to the hospital for delivery. The protocol excluded (1) mothers with more than four previous births, (2) mothers less than 18 years of age, (3) mothers who had resided fewer than 5 years in their district, and (4) mothers with a major illness during pregnancy.

Following birth, we also excluded mothers whose infants had severe birth defects. Follow-up occurred at 6, 16, 45and at 72 months. Data on breastfeeding were collected by a qualified nurse during follow-up examinations. Concentrations of 15 PCB congeners (IUPAC No. 28, 52, 101, 123, 118, 114, 153, 105, 138, 167, 156, 157, 180, 170 and 189) in the umbilical cord and child serum were determined as described elsewhere (Kocan et al., 2004; Conka et al., 2005). All analytical measurements were carried out at the National Reference Centre for Dioxins and Related Compounds (Department of Toxic Organic Pollutants, Slovak Medical University) which has been certified by the Slovak National Accreditation Service (ISO/IEC 17 025:2005, certification No. S-111) and regularly participates in interlaboratory studies and proficiency tests on dioxins and PCBs in food and feed. Control charts were plotted for QC samples, blanks, and verification calibration standards to check accuracy, precision, and reliability of the analytical process. Only samples with PCB concentrations ≥LOD were taken into account. We report lipid adjusted concentrations. We estimated total serum lipids using the enzymatic summation method (Akins et al., 1989). We fitted a concentration-time function (Trnovec et al., 2011) to serum PCB 153 concentration data of each child and calculated PCB 153 serum concentration at time of weaning. The serum concentration values at the time of weaning for each particular child were approximated by polynomial function of degree 2 of the form $y = ax^2 + bx + c$.

Statistical calculations were carried out with statistics program SPSS 16, Softonic International S.L.

3. Results

We have used data on PCB 153 serum concentration measured in time intervals 0, 6, 16, 45 and 72 months after delivery (Table 1) for determination of the PCB 153 serum concentration at the time of weaning.

We present the descriptive statistics on parameters of the function approximating the concentration time course of each child in Table 2.

In Fig. 1A we show the over 3 orders of magnitude variability of PCB 153 cord serum concentration. It is reflecting variation of PCB 153 in maternal blood and rate of placental transfer of PCB. In order to eliminate the projection of this variability to postnatal period we have adjusted the current postnatal serum concentration values of infants to cord serum PCB 153 concentration as C_{tbf}/C_0. In Fig. 1B we present a frequency diagram of time of breastfeeding.

We show the resulting serum concentration values at the time of weaning for each particular child in Fig. 2A. We have examined approximation of this relationship by several functions and obtained optimal fit for polynomial approximation of degree 2. Replacing C_{tbf}/C_0 values by arithmetic means calculated for every month (while at least 2 values were available) markedly improved the fit (Fig. 2B).

When calculating the values of the model parameters by iterative Monte-Carlo simulation procedure, we observed that the model was very sensitive to improbable data reported by mothers on duration of breastfeeding. The model assumes continuous uptake of PCB at a constant rate and an instantaneous stop of uptake, i.e. abrupt finishing of breastfeeding. In real life however the rate of breastfeeding diminishes gradually. Abraham (2002) obviously was aware of this fact when modeling exposure and introduced the "equivalent to duration of exclusive breastfeeding" in his study. The model also does not account for reuptakes of PCB 153, however breast milk scarcely can be a source of reuptakes. These can occur with normal food, but in infancy may be rare. If the child is living in an area highly polluted by PCBs and after weaning is fed by food produced locally (Sonneborn et al., 2008a, 2008b), reuptakes at adolescent age can occur, as we have shown previously (Wimmerová et al., 2011). A slight modification of the weaning time usually improved the fit. Consequently we consider our t_{bf} values equivalents to duration of exclusive breastfeeding as introduced earlier by Abraham (2002).

4. Discussion

We are characterizing the relationship between PCB 153 serum level of infants and duration of breastfeeding as a continuous nonlinear function. It is based on serial sampling in 305 infants. For each 5 concentration measurements spaced in time were available and information on length of lactation. The function for averaged data in Fig. 2B reliably

predicts burden of PCB 153 of breastfed infants at time of weaning which is important for dose-outcome relationships.

The concentration time course, C_t, in each child starts from C_0 which largely varies as we have shown in Fig. 1A. By adjustment of current concentrations, C_{tbf}, to cord blood serum concentration, C_0, all individual time courses start from a value of 1, as for time zero $C_t=C_0$. Such adjustment eliminates dependence of the infant's body burden of PCB 153 on a variable represented by maternal body burden which depends on mother's age, changes in body composition and exposures early in life (Lignell et al., 2011). Similar presentation as in Fig. 2 can be found in literature (Tohyama et al., 2011; Abraham, 2002), however without adjustment to C_0. Adjustment to C_0 and fit by a polynomial instead a linear function improved the outcome.

Curves in Fig. 2 show that C_{tbf}/C_0 peaked at extremely long nursing periods at values between 4 and 5 which is in agreement with 4.5 published for children at 42 months post partum (Lanting et al., 1998). The flattening and downward trend may result from approaching equilibrium between intake and elimination and distribution of PCBs into an increasing pool of lipids as child grows.

To predict the absolute PCB 153 concentration C_{tbf} from C_{tbf}/C_0 values we need to know C_0. An advantage for obtaining C_0 is however that when expressed on a lipid basis, maternal plasma, cord plasma, and milk concentrations of PCBs are strongly intercorrelated, indicating that PCB concentration in any of these biologic media is a good indicator of C_0 (Ayotte et al., 2003). We confirmed that conversion of PCB data from one matrix to another is feasible (Govarts et al., 2012). An advantage of our model is that it predicts infant's PCB concentration over whole nursing period contrary to published single time point predictions (Ayotte et al., 2003).

Conclusion

Our aim was to improve assessment of exposure to PCBs resulting from breastfeeding and to upgrade the prediction of exposure based on known time of nursing. In a cohort of 305 children we determined PCB 153 serum concentration at the time of weaning and described this relationship with a mathematical function enabling reliable prediction of PCB exposure in infants for scientific purposes or risk analysis.

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Highlights

- Breastfed infants are at risk of exposure to considerable amounts of PCBs.
- Determination of perinatal exposures to PCB 153 is essential for risk assessment.
- We determined PCB 153 serum concentration at time weaning of breastfed infants.
- We reliably predicted exposure to PCB 153 of breastfed infants.

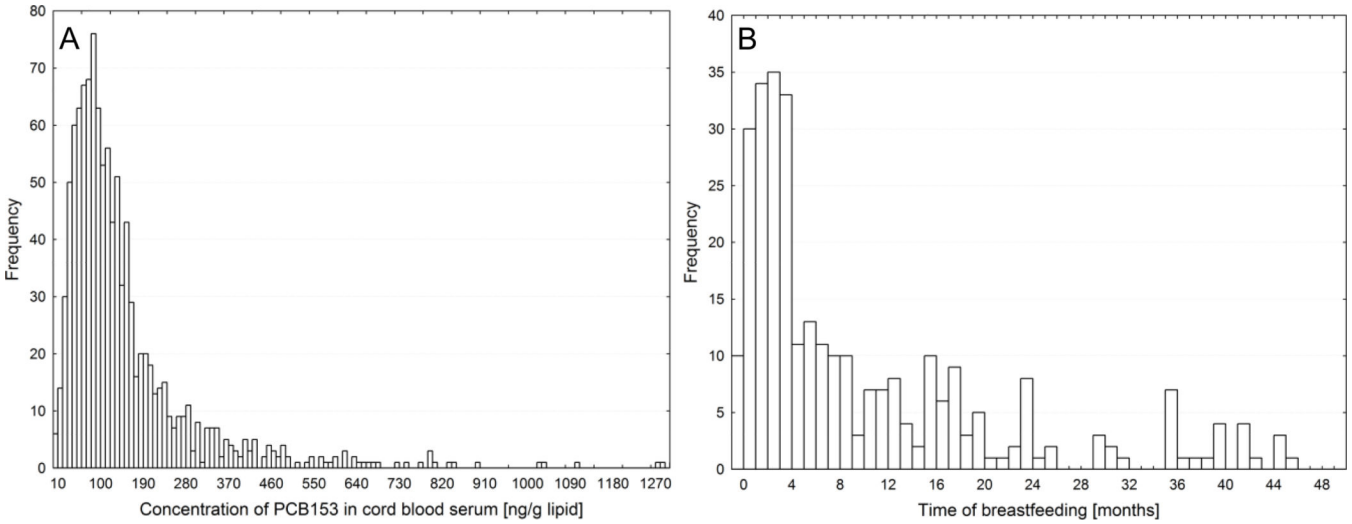

Fig. 1.

Frequency diagram of PCB 153 concentration, ng/g lipid, in cord blood serum, C_0, of 1082 children of which are 305 children of the current cohort a subgroup (A). Frequency diagram of duration of breastfeeding, t_{bf}, of mothers of the current cohort (B).

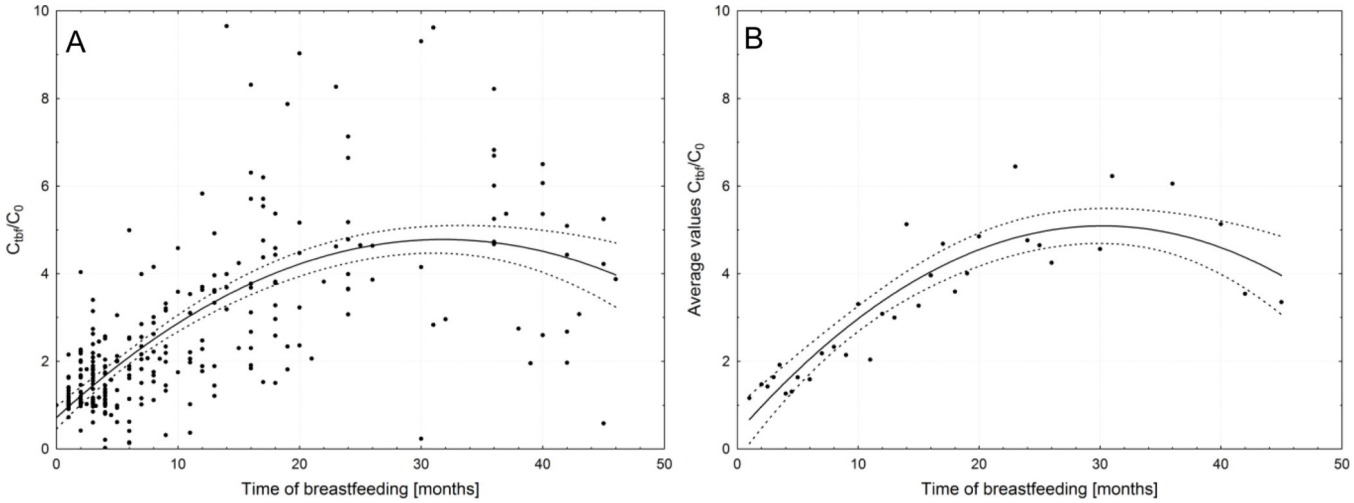

Fig. 2.

Computed serum concentrations of PCB 153 adjusted to cord serum concentrations, C_{tbf}/C_0, at the time of weaning, t_{bf}. Each point represents a child and the curve is the best fit of a polynomial function of degree 2 ($C_{tbf}/C_0=0.596+0.278t-0.0047t^2$) and its 95% confidence interval (A). Averaged data ($C_{tbf}/C_0=0.156+0.334t-0.0059t^2$) (B).

Table 1

Data on concentration of PCB 153 in blood serum of infants in ng/g serum lipids at various time intervals after birth and on equivalents to duration of exclusive breastfeeding in months.

	Time after birth (Months)	Concentration of PCB 153				
		Mean	SD	Minimal value	Median	Maximal value
Child serum	0	185.65	170.5	14.1	137.45	1373.7
	6	228.21	312.54	1.69	141.17	2640.76
	16	268.86	402.75	0.52	137.91	3024.45
	45	227.71	325.63	3.2	116.49	2749.41
	72	93.84	133.56	4.4	48.36	1011.62
Equivalent to duration of exclusive breastfeeding		10.27	11.13	0	5	46

Table 2

Parameter values of the model applied to postnatal PCB 153 serum concentration values in 305 infants of our cohort. $C_{bf,\infty}$ is a hypothetical limit value of PCB 153 concentration increase for t and time of breastfeeding, t_{bf}, converging to infinity and $C_{f,\infty}$ is a hypothetical limit value of PCB 153 concentration increase for t converging to infinity and t_{bf} converging to zero and MT is mean time, months, of PCB 153 in the body of infants. Area under the curve from time zero to 72 months, AUC, is in ng.months.(g lipids)$^{-1}$. C_0 is concentration in cord blood serum.

	Mean	SD	Minimal value	Median	Maximal value
MT	66.52	193.51	0.003	5.19	2330
AUC	15150.7	20994.9	824	8660	151000
$C_{bf,\infty}$	1543.74	4929.44	0.5	395.72	75000
$C_{bf,\infty}/C_0$	10.37	25.7	0.008	2.74	265.63
$C_{f,\infty}$	1009.26	3984.32	3.18	136.93	62000
$C_{f,\infty}/C_0$	7.02	20.49	0.05	1.03	214.45
$C_{f,\infty}/C_{0,\infty}$	0.58	1.59	0.03	0.42	26.82
C_t/C_0	2.41	1.8	0.02	1.82	9.65

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