Time trend of dioxins in breast milk in Sweden 1996-2004

Sanna Lignell[1], Anders W Glynn[1], Per Ola Darnerud[1], Marie Aune[1], Rickard Bjerselius[1], Bert Baumann[2], Sven Cnattingius[3]

[1]Swedish National Food Administration
[2]National Institute for Public Health and the Environment
[3]Department of Medical Epidemiology, Karolinska Institutet

Introduction

Breast milk has been used for monitoring of human body burdens of persistent organic pollutants (POPs), eg. polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs) and dibenzofurans (PCDFs), for many decades. POP levels in breast milk reflect the long-term exposure of the individual mother and also estimates the body burden during pregnancy, when the critical exposure of the fetus occurs. In Sweden (Stockholm), time trends of PCBs and PCDD/DFs have been studied since the early 1970s[1]. During the period 1972 to 1997, the total PCB levels declined 3.3-fold and the levels of total toxic equivalents (total TEQ) of dioxin-like PCBs and PCDD/DFs declined 3.2-fold. In those studies, time trends were assessed on pooled milk samples, making it impossible to account for factors influencing the PCB and PCDD/DF levels, such as age of the women and parity[2,3].

In 1996, the Swedish National Food Administration (NFA) started a time trend study of POPs in breast milk from primiparous women living in Uppsala County. Analyses of the compounds have been made on individual samples, making it possible to statistically adjust for lifestyle/medical factors affecting the levels in breast milk. Results from the analyses of PCDD/DFs in 77 breast milk samples from 1996 to 2001 have been reported earlier, and in that report no significant negative association was found between sampling year and age-adjusted PCDD/DF TEQ concentrations[4]. Additional breast milk samples have now been analysed, and we present results from the analyses of PCDD/DFs in a total of 154 breast milk samples collected from 1996 to 2004.

Material and methods

Primiparous women from the general population in Uppsala County were recruited from January 1996 to May 1999 (N=211), from April 2000 to March 2001 (N=31), from March 2002 to February 2003 (N=31) and from January to December 2004 (N=32). The mothers sampled their milk at home during the third week after delivery, using a manual breast pump and/or a passive breast milk sampler. Milk was sampled both in the beginning and at the end of each breast-feeding session. The goal was to sample 500 mL milk from each mother during 7 days of sampling. During the sampling week, the breast milk was stored in acetone-washed bottles in the home freezer. Newly sampled milk was poured on top of the frozen milk. Data on age, weight, lifestyle etc. of the participating mothers were obtained via questionnaires.

The PCDD/DFs (17 congeners) were analysed at the National Institute of Public Health and Environment (RIVM), the Netherlands, using methods described in Glynn et al.[2]. A total of 154 samples from mothers who were all Swedish by birth were analysed. Concentrations of the individual PCDD/DF congeners below LOQ were set to ½LOQ in the calculation of PCDD/DF TEQ. Statistical analysis was performed using simple and multiple linear regression analysis.

Results and discussion

A compilation of basic data for the participating women is given in Table 1.

Table 1. Basic data for the mothers participating in the study (N=154).

	N	Mean	Median	Range
Age at the milk sampling occasion (years)	154	28.7	29.0	21.0-37.4
Body Mass Index (BMI) before pregnancy (kg/m^2)	153[b]	22.9	22.2	16.2-37.7
Weight gain during pregnancy (% of initial wt/week)	153[b]	0.64	0.60	0.03-1.54
Weight reduction from delivery to sampling[a] (%)	147[b]	9.46	9.23	1.2-19.1
Birth weight of the child (g)	153[b]	3603	3510	2610-5001

[a]Weight reduction minus birth weight of the child in % of weight just before delivery.
[b]Some of the mothers did not answer the questionnaire completely.

The median PCDD/DF TEQ concentration was 8.0 pg/g lipid with a 6.5-fold difference between the highest and lowest concentration (Table 2). Simple linear regression showed that the concentration of PCDD/DF TEQ increased 0.41-0.53 pg/year of age of the participating women ($p<0.001$). Simple linear regression also showed that the concentration of PCDD/DF TEQ decreased with increased weight gain during pregnancy (regression coeff. –3.59, $p=0.003$). No significant relationships between PCDD/DF TEQ concentrations and Body Mass Index (BMI) before pregnancy or weight reduction from delivery to sampling could be observed.

Table 2. PCDD/DF concentrations (pg/g lipid) in breast milk from primiparous women living in Uppsala County 1996-2004. Concentrations of the individual PCDD/DF congeners below LOQ were set to ½LOQ in the calculation of PCDD/DF TEQ.

	N	Mean ± SD	Median (min-max)	1st quartile	3rd quartile
PCDD/DF TEQ	154	8.6 ± 3.3	8.0 (3.56-23.1)	6.3	10.8

Multiple linear regression analysis showed that there was a negative association between sampling year and concentration of PCDD/DF TEQ (ln-transformed data) (Table 3, Figure 1). Age and weight gain during pregnancy were included as explanatory variables in the regression model. The results indicate that the average concentration of PCDD/DF TEQ has declined 5.9-7.5%/year, among primiparous women from Uppsala County, during the period 1996 to 2004. Based on these results the half-life of PCDD/DF TEQ was estimated to 10 years. In our earlier study, from 1996 to 2001, no significant negative association was found between sampling year and age-adjusted PCDD/DF TEQ concentrations[4]. Earlier studies in the Stockholm/Uppsala area have shown declining PCB and dioxin concentrations in breast milk during the time period 1972 to 1997[1,3]. These studies have, however, not been able to account for differences in age etc. of the participating women. The estimated half-lives for PCDDs and PCDFs in the Stockholm study were 15 and 11 years respectively, which is slightly longer than the half-life for PCDD/DF TEQ estimated in our study.

Table 3. Time trend (1996-2004) of PCDD/DF TEQ in breast milk from primiparous women living in Uppsala County, Sweden (adjusted for age and weight gain during pregnancy).

	N	Regression coefficient[a] ± SD	R^2	P
PCDD/DF TEQ	153[b]	-0.0694 ± 0.0085	55%	<0.001

[a]Concentrations ln-transformed.
[b]Mothers who did not answer the questionnaire completely (N=1) were excluded.

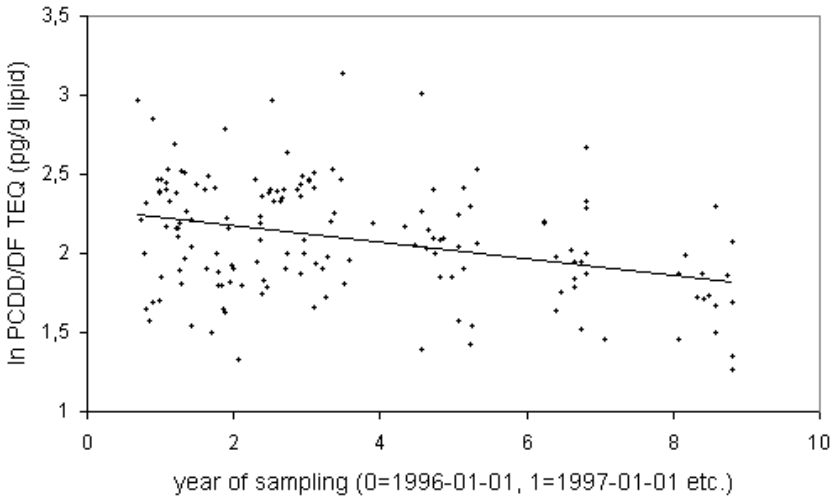

Figure 1. Time trend in breast milk concentrations (N=154) of PCDD/DF TEQ in primiparous women from Uppsala County, Sweden from 1996 to 2004. A significant negative association between sampling year and concentration of PCDD/DF TEQ was shown in a multiple linear regression including age and weight gain during pregnancy as explanatory variables.

In this study we were able to account for life-style/medical factors that influence breast milk levels of POPs. For instance, only primiparous women were recruited. It is well known that breast-feeding is a major pathway of POP excretion in women, and consequently the POP levels are usually higher in breast milk sampled after the first child is born than in breast milk sampled after subsequent deliveries[3]. Since POPs accumulate in the body it is also important to account for the age of the participating women in this type of studies. In our study, the concentrations were age-adjusted in the calculation of half-life, thus taking care of possible differences in average age of the women between sampling years. The concentrations were also adjusted for weight gain during pregnancy in the calculation of half-life. The negative association between weight gain and PCDD/DF TEQ levels was probably due to a diluting effect of the extra fat tissue.

Acknowledgements

Appreciation is expressed to the participating women, to midwives Irma Häggbom, Ragnhild Cnattingius, Margareta Aveskog, Ingela Wessén, Astrid Bengtson, Toni Eriksson, Ann Rydberg and Marianne Leimar, who assisted in recruitment, interviewing, and sample collection. Laboratory technicians Ingalill Gadhasson, Lotta Larsson and Elvy Netzel are appreciated for technical assistance.

References

1. Norén K. and Meironyté D. (2000) Chemosphere 40: 1111-1123.

2. Glynn A., Atuma S., Aune M., Darnerud P.O. and Cnattingius S. (2001) Environ. Res. 86 (3):217-228.

3. Vaz R., Slorach S.A. and Hofvander Y. (1993) Food Addit. Contam. 10: 407-418

4. Glynn A.W., Darnerud P.O., Aune M., Bjerselius R., Baumann B. and Cnattingius S. (2003) Organohalogen compounds 64:21-24.